THE NATURE OF RADIOACTIVE FALL-OUT AND ITS EFFECTS ON MAN

HEARINGS

BEFORE THE

SPECIAL SUBCOMMITTEE ON RADIATION

OF THE

JOINT COMMITTEE ON ATOMIC ENERGY

CONGRESS OF THE UNITED STATES

EIGHTY-FIFTH CONGRESS

FIRST SESSION

ON

THE NATURE OF RADIOACTIVE FALLOUT AND ITS EFFECTS ON MAN

JUNE 4, 5, 6, AND 7, 1957

PART 2

Printed for the use of the Joint Committee on Atomic Energy

UNITED STATES
GOVERNMENT PRINTING OFFICE
WASHINGTON : 1957

STATEMENT OF DR. HERMANN J. MULLER, PROFESSOR OF ZOOLOGY, UNIVERSITY OF INDIANA [4]

Dr. MULLER. Thank you, Mr. Chairman.

The subject of the genetic effects of radiation is, in my opinion, so closely bound up with that of the effects on the exposed individual himself that I feel it necessary, to begin with, to touch on that subject first.

I feel this is especially necessary because there has been a curious official silence concerning findings showing that the main damage to the exposed individuals themselves by small or moderate exposures to radioactive substances or X-rays consists of an insidious weakening of the body's resistance to the onset of infirmities and diseases of all kinds, expressing itself in a shortening of the length of life, and also consists in a long delayed production of certain specific disorders of which the most important are leukemia and some other malignant conditions.

Still less publicized has been the increasing evidence that the amount of these effects is simply proportionate to the total dose of radiation received, even when this has been given in tiny bits scattered over long periods. That is evidence for the conclusion that there is no threshold, some of which evidence you heard presented by Dr. Lewis.

I may say that I first heard of this principle of the shortening of life in amounts proportionate to the total dose received, no matter in how many small bits, in a very scholarly address given by Dr. Robert D. Boche at Oak Ridge in April 1948, and again at the Argonne Laboratories in November 1948. These were not classified meetings. The principle that he explained to them was based on work done by a number of different investigators during the war, working mainly at Rochester, N. Y., under the Manhattan project.

I mentioned these conclusions of Boche in a series of lectures given under the auspices of the Society for Sigma Xi, that were published 2 years later. But I was unable to refer to Dr. Boche's work except as addresses because I waited in vain to see publications along those lines.

Representative HOLIFIELD. Where were these addresses given?

Dr. MULLER. One was at Oak Ridge and the other at Argonne. They were open meetings. That is to say, it was not classified. They

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were not published and they were not open to the press so far as I know, so I only knew about the matter verbally.

Representative HOLIFIELD. Were they in the form of prepared addresses?

Dr. MULLER. Prepared addresses, and with charts of data; yes.

Representative HOLIFIELD. Who did you expect to print those?

Dr. MULLER. I expected he would have an article on the subject. Later I found that there was a classified article of his in 1946 on the subject. It was later declassified, but I am not sure when. It did not get out into the general literature. It was not spoken about to any extent. In fact, although I do not know whether it had any relation to that silence or not, there was an attempt made to prevent the publication of that part of my own lecture which contained this material. No special reason was given. I do not want to go into the details of that because it may have been accidental, but it is a very curious incident.

Representative HOLIFIELD. You said there was an attempt made?

Dr. MULLER. The editor had agreed to publish the whole article. It was presented in 2 parts because it was too long to go in as 1 article. Both parts were to have been published. After the one part had been published I was told that they did not want to publish the other part, that that was enough. The first part concerned itself with the effects on future generations and the second part with the effects on the exposed generation itself.

Representative COLE. Mr. Chairman, would Dr. Muller give the time when this incident occurred?

Dr. MULLER. The lectures were given in 1948. They were published first in 1950. So that occurred in 1950.

Representative VAN ZANDT. Dr. Muller, did the thinking at that time fit in with the problem of today?

Dr. MULLER. Yes. It was not as acute because we did not have hydrogen bombs yet.

Representative VAN ZANDT. Were there any predictions in those papers?

Dr. MULLER. I called attention to these points; yes. We did not know much about fallout yet.

Representative HOLIFIELD. Dr. Muller, what type of a meeting was this? Who set the meeting up, and where was it?

Dr. MULLER. One of them at Oak Ridge was set up by the Director of Research, Dr. Hollaender, a very eminent biologist and geneticist. The other was called under the auspices of the Argonne Laboratory on the problem of the effects of low doses of radiation.

There were many interesting papers there. For example, one which showed the lessening of the number of white blood cells in workers at Los Alamos who had received only 25 r. of radiation over the whole year. Nevertheless, by taking accurate statistics of the whole group it was possible to show the effect. One would not have been able to see it in one individual because it fluctuates too much.

Representative COLE. Mr. Chairman, I am curious to know, Dr. Muller, is it your feeling that there was a deliberate attempt on the part of the responsible officials of the Government, whether of the Atomic Energy Commission or the Manhattan Engineering District, to suppress the expression of your views on this subject?

Dr. MULLER. I would not like to express an opinion about that. It may have been a coincidence. But I was very surprised that no one else said anything about the matter in any semipopular or popular publication. This was a semipopular publication.

Representative VAN ZANDT. Dr. Muller, do you feel that had the papers been made public at that time that it would have eased this acute problem we are faced with today?

Dr. MULLER. Yes. I think that any such knowledge is to the good because the public should be prepared to know the facts and if they find out later that something has been withheld from them—I will touch on that later—they are apt to exaggerate the importance of the facts. I think that there was a feeling probably amongst many people who felt that these facts needed further verification, that it was unwise to let the public know about them until they were proved up to the hilt.

To me it seemed that the main principles were already far enough established to warrant a public airing, since if true, they were of such grave concern.

Senator ANDERSON. Doctor, this morning there was some reference made to Russia, and it was suggested that one of the witnesses testify about the situation in Russia. Here we have the situation in this country today where the foremost geneticists of the country are testifying before an open hearing. Is that the situation in Russia, or are geneticists in somewhat different circumstances over there?

Dr. MULLER. There are few geneticists left. I would rather postpone questions on Russia until we get through this because we might go on indefinitely. I think we can draw lessons from Russia.

Senator ANDERSON. I knew you had worked in Russia.

Dr. MULLER. Yes.

Senator ANDERSON. And therefore have some opinions. I hope you give them when you finish this afternoon.

Dr. MULLER. I would be happy to answer to the best of my ability any such questions as you might have.

These facts, which I think Dr. Hardin Jones and Dr. Russell will take up in their talks, about the shortening of the length of life seems to me to be extremely important. I think it can be shown that they and the long delayed malignancies such as leukemia constitute by far the greatest damage of all the effects of radiation on the exposed individual himself. That is, of moderate and small doses. I am not speaking of the radiation sickness and death that occurs from very high exposures such as direct atomic bombing.

Therefore, I thought it was very curious that these effects were not discussed more and were not in fact investigated more. It seemed to me if there was an uncertainty then it should have been pushed to find out is it true or not.

We did finally in 1954 have papers on this work published in declassified form in a highly technical volume edited by Dr. Blair almost 10 years later. Since then the effects have been talked about to some extent. They were mentioned in the Pathology Committee report of the National Academy of Sciencies. So far as I can remember in the National Committee on Radiation Protection—I happened to belong to its Subcommittee on External Dose for some years—there was very little mention of them. I doubt very much that they were gen-

erally realized by workers in the field of radiology, for example, or by biologists in general.

In respect to the fact that probably there is no threshold, that these effects are proportional to the dose, in this respect these effects of radiation—and also the leukemia—on the exposed individual himself resemble those produced by the radiation in weakening descendants.

You have heard Dr. Glass and Dr. Crow say that geneticists are convinced that there is no threshold for the genetic effects and that others, too, now accept that principle for the genetic effects.

If this is true of these other effects, and it is certainly time we knew whether it was—I think the evidence is convincing that it is—then this important resemblance between the effects on later generations and on the exposed generation is probably not an accidental resemblance. For there is growing reason to infer that this shortening of life and the other long delayed damage done to an exposed individual have their basis in damage done to the genetic material—the chromosomes and their contained genes—of the body's ordinary cells, those of the blood, skin, glands, and so forth, similar to the damage done in his reproductive cells that is passed on to later generations.

In the body's own cells, however, we have good reason to infer that more of the damage that becomes expressed is done by breaks of chromosomes and in the reproductive cells by mutations of genes.

Let me use the blackboard a moment.

Here is a cell, and there is its nucleus, and in it there are these long threadlike bodies which can become condensed into sausage-shaped forms, called chromosomes, that consist essentially of strings of minute particles called genes, each of which, as Dr. Glass explained to you, has some specific effect in furthering the functioning of the body.

Radiation has two main kinds of effects on this genetic material. Let me again say that every cell of the trillions of cells in the body has this genetic material the same as the reproductive cells, and radiation can affect the genetic material in the body cells the same as in the reproductive cells.

There are two main kinds of effects on the genetic material. One is a break in a chromosome. Two, a change in the inner composition or arrangement of parts of the gene itself.

The breaks in the chromosomes have more effect on the exposed individual himself when they happen in the somatic cells for this reason. If this cell in the diagram is one that is going to later reproduce itself in the body to form more cells, or to multiply, as our skin cells must—as skin is always sloughing off and has to be replenished by multiplication of the cells in the lower part of the skin, and it is the same with many other parts of the body—then these chromosomes have to reproduce themselves, as Dr. Glass explained, forming duplicates of themselves. Then they are drawn apart by the process of cell division or mitosis, forming 2 groups of chromosomes in the 2 daughter cells. But if the chromosome is broken, it may kill the whole cell at the time or shortly after division by this mechanism, or it may result in very abnormal cells.

There is a point on each chromosome, which becomes attached to a fiber that pulls the 1 daughter chromosome to 1 pole and the other to the other pole, so that when you get the cell dividing into 2 daughter cells, one daughter cell gets one representative and the other gets the other representative of that duplication process, and they thereby come

to have like material. The chromosome gets pulled at that particular point. But the part beyond the break won't be pulled in because it has nothing to be pulled by. It gets left behind, and the cell is left without all those genes.

Moreover, where the chromosomes have broken, there is a sort of stickiness which causes them to join together at their raw ends if they happen to touch, which is very often the case, and then, when they get pulled apart, they form what we call a bridge between the two cells. They can't, in that case, get apart. That chromosome bridge, then, connects the two cells, which often die in consequence. Therefore, as a result of the chromosome break, we often have a cell death.

Dr. Puck, of Denver, studying human cells in tissue cultures, has found that, with doses of radiation given at a high dose rate, it takes only about 90 r. to cause the death of about 50 percent of the cells. In other words, that happens to about 50 percent of the cells in the tissue culture. It will only happen to cells which attempt to undergo division.

Your brain cells, your kidney cells, as Dr. Warren said yesterday, are not affected much by radiation. On this interpretation the reason is easy to see, because they have stopped dividing for life. You have the same cells of these kinds for life, whereas your skin cells, the cells of the intestine, et cetera, have to keep replenishing themselves by division. It is only when they come to replenish themselves by division that these tangles take place that result from the breaks in the chromosomes.

On that basis it is easy to understand the rule that Dr. Warren referred to yesterday, sometimes known as the law of Bergonié and Tribondeau, established about 1906, that it is the tissues with rapidly dividing cells that are the most damaged by radiation. The damage does not take place in the cell until division occurs. When that happens in a germ cell, those cells generally die and seldom take part in the production of offspring. That is why this is not important for the later generations. But it is important for the generation itself. It takes place both in the germ cells and body cells, but it is in the body cells that the main damage of this kind is done.

I will give you an illustration of that. In the people that were bombed a long time ago at Hiroshima you can see a lot of little opaque points in many of them in the lens of the eye where evidently cells have been badly damaged. That damage did not appear until much later when the cell probably undertook to divide. The reason you can see it there is because the lens is a transparent tissue. I think there is reason to believe that this happens in all the tissues of the body that contain cells that are subject to division. These tissues would, therefore, be weakened.

It is true that other undamaged cells would tend to replenish the damaged places. But that replenishment or regeneration, it is to be expected, will not be complete and perfect. Therefore, there is a certain amount of damage left. That damage, you can see, would be a generalized damage all over the body, wherever there are these dividing tissues. Therefore, it would be expressed as a weakening of resistance to disease and infirmities of all kinds, somewhat like what occurs in aging, since then, also, our resistance to bodily ills decreases as the functioning of the cells or tissues weakens.

I do not say by any means that is the proved explanation, though we do know this phenomenon of chromosome breakage occurs, and, to me, having examined the evidence, it seems by far the most reasonable explanation. That is, the bodily damage in general is to be explained in this manner rather than by some damage to the other materials of the cells.

There is a good deal of other evidence showing that it is this damage to the genetic material, to chromosomes and genes, that is by far the most important damage, and that you have to have the radiation strike at or in the close neighborhood of the chromosome or gene before you can damage it. For example, experiments done in Chicago by Zirkle and Bloom show that when they were able to direct a minute beam of radiation (protons) through the cell, it was only when they actually hit the chromosome or right close to it that you then got chromosomal damage. If you hit right there, when the cell divided you had the bridges form.

Other experiments similarly show that if you only hit the rest of the protoplasm you do relatively minor damage compared to what happens when you hit the chromosomes or genes.

You can readily see, I think, that since we know through experiments in geentics that the frequency of these breaks, like the frequency of the mutations of the genes, is linearly proportional to the dose of radiation used, no matter in how small bits it is divided, then you might expect a derived effect, such as the decreased resistance to disease and consequent shortening of the life span also to be linearly proportional.

It is true that at high dose rates of radiation you sometimes have two chromosomal breaks near together and then you can get entanglements which would not happen if you have low dose rates. At low dose rates you therefore expect the effect to be proportional but at the high dose rates to go up even more steeply.

I think it is also very probable that even the very pronounced effects of heavy exposure, such as you find in radiation sickness, such as the nausea, the drastic lowering of the count of white blood cells, the bleeding internally, are also due to this genetic damage to the somatic cells.

For example, in a very careful investigation, Dr. Quastler showed that the intestinal injury that leads to death after high doses is caused by the failure on the part of the cells of the intestine that normally replenish the rest every 3 or 4 days to carry out this task. Owing to being so badly damaged, they fail to survive the process of cell division in normal condition.

These effects of heavy doses do have thresholds, not because there is a threshold in the production of chromosome breaks, but only because you do not see clinical symptoms unless you have damaged a certain number of cells.

If this point of view is correct, then I think it is to be expected that even the damage to the exposed individual could be better investigated by people who have the genetic point of view about it. They would be the ones who would be more likely to look for an effect that has no threshold. I think it is not at all surprising, therefore, in view of the probable mechanism of these effects, that it has taken geneticists, notably Boche, in the case of life span, and Lewis, in the case of the

effect of leukemia—both, by the way, *Drosophila* geneticists originally—to uncover the evidence for the cumulative nature of the damage to exposed individuals. Moreover, I think the research should be continued along those lines.

Doctor Friedell said yesterday that the important thing to know is the mechanism involved. I do not say we know it. But I think we have more than a pretty good hunch. And we have to follow these hunches, even if they lead to conclusions that are distasteful to some people, such as that there is no threshold. But if we calculate (as Dr. Hardin Jones will calculate with you) the amount of effect of a given dose on the length of life, we find that the dose which is now the maximum permissible dose for occupationally exposed workers, namely, 50 r per 10 years, would lead in 40 years of their work to 200 r. and would thereby deprive them—we may want to change this estimate; we can't now say exactly—of some 4 years of their life. Perhaps 1 year of their life lost for each 4 years they work, or something like that.

My main point here is that if there is no threshold, then the loss is of a sizable amount and we had better pretty soon find out how much. We meanwhile have to act on the supposition that it exists. Nevertheless, this effect on the length of life of the exposed generation is not as great as the effect in damaging future generations.

Representative HOLIFIELD. Why is that, Doctor? Will you explain that?

Dr. MULLER. Yes. Let me now speak of the damage to future generations. I said that not much of it was caused by the chromosome breaks but most of it by the mutations of the genes. The reason the mutations of the genes do not cause much damage usually to the exposed individual is because most of them are what we call recessive. There is a normal gene from the other parent that has a dominating effect, although it may very well be the case that in leukemia we have a dominant gene. But mutant genes that are decidedly dominant are not the usual ones.

As for later generations, the chromosome breakage cases are largely weeded out by the cells dying before they get to the next generation. The mutations of the genes, however, persist and are handed down as mutant genes. These mutant genes also are usually recessive, as Dr. Glass explained. The person usually gets a normal gene from the other parent and that has the dominating effect. But the dominance is not quite complete usually, and that slight deviation from completeness is very important.

Suppose it only reduces by 5 percent the chance of an individual surviving to maturity; that is a chance of death of 1 in 20. It does so by handicapping him in some way. It is usually a slight handicap that he hardly realizes is there. He takes it in his stride because he has had it perhaps from birth, though it may not have expressed itself always, and it is mixed in with his other infirmities. All of us have some. No one is perfect because there is no such thing. But by it his biological survivability is reduced by just that much, and he hands that weakness on down to the next generation, and after awhile it will take its toll by happening to come in a combination of circumstances where it will kill or prevent reproduction. So the thing will finally die out.

As Dr. Crow explained, if it gives a chance of 1 in 20 of causing death in 1 individual, it tends to pass down to 20 individuals before it takes its toll. So it hampers correspondingly more persons than a gene that killed outright. Therefore, as Dr. Crow explained, the slight mutation is as bad in the end as the mutation with the big effect. Moreover, we are all of us full of these defects that come from the past. A hundred things, each of which does a one-hundredth of as much harm, are together as bad as one thing that does that much harm.

Representative HOLIFIELD. Before you leave that, is it not true that if a general population receives a dose of radiation, as would occur in the general raising of the rate in the atmosphere, that there would be a greater chance for the mating of recessive genes?

Dr. MULLER. If both people had been exposed, no, the increase in the chance would be insignificant, contrary to a common misconception. Because the chance is so slight that it should have been the same gene that was effected in both parents. There are more than 10,000 genes, we believe, and if each parent had an affected gene, therefore, the chance would be only 1 in 10,000. So that is virtually ruled out.

Senator ANDERSON. May I go back one step? Did I understand you to say that if, for example, the exposure was 50 r. every 10 years, and a man worked for 40 years, making a total of 200 r., his life might be shortened as much as 1 year for every 4 years of work?

Dr. MULLER. No; I am sorry. I made a mistake. I means 1 for every 10. Dr. Hardin Jones will give you the latest information.

Senator ANDERSON. You said 1 for every 4.

Dr. MULLER. Yes. I was thinking of 4 times 10, and I got the 4 instead of 10.

Senator ANDERSON. So that would be 4 years shortening.

Dr. MULLER. Yes. That would be the provisional estimate I made a long time ago and we may have better data now. I think this is probably a conservative estimate.

Dr. JONES. A very reasonable estimate.

Senator ANDERSON. Thank you.

Dr. MULLER. These effects then, being slight, are usually not recognized as such. Certainly you could not tell which mutation was caused by radiation, if radiation had been received by the parent, and which was not. The induced mutations are like those already existing in the population but are added to them. The only way you can tell they have been produced is by means of a very exact statistical study on large groups, comparing those that have been irradiated and those that have not.

Through work on the fruitflies where we have the most exact knowledge to date, unless Dr. Russell has more exact knowledge on mice now, we can get a kind of minimum estimate of the amount of damage to the children by a given amount of irradition of the parents. Although there would not be time to show you here the way the calculations are done—and they do have a considerable error—I think it is possible to show that the amount of damage to the offspring of parents that had received a certain amount of radiation to their whole body, in the case of fruitflies, would be something like the amount of damage in the parents themselves.

A comparison, however, would show that, when, X-rays or gamma rays were used on fruitflies, the damage to the offspring would be some-

by what I said before when I said the damage to future generations is greater than to the parents. There is no real contradiction because we are here referring only to the first generation of offspring. But they hand the damage down to their offspring, and so on.

As Dr. Crow explained to you, the damage does not die out to half its value for probably scores of generations. So you have to multiply this damage to the first generation of offspring by scores, maybe by 50, to find out what the total damage to future generations is of just the 1 exposure to 1 generation. When you have done that you have obtained a figure of damage to future generations that is far greater than what is done to the exposed generation itself.

I think most people would not be impressed with the weaknesses caused in future generations, even though future generations would feel them. Therefore, it is my contention that it is a very good thing that people's own life span is shortened; that is, that there is a demonstrable effect on the generation itself that is exposed, because they will take notice of this effect on themselves, if they are allowed to know it. They will then take precautions that will save future generations from a lot more damage than it saves themselves.

The prolonged official reluctance, at least until a year or two ago, to give information in popular form regarding these major types of radiation damage, that in the exposed individual himself expressed in the shortening of life and long delayed malignancies and that expressed in the descendants, and the reluctance to give information regarding the conclusion of some of those who have worked most directly in the field that even the tiniest doses add up accurately to determine the amount of these effects without any threshold, has, I think, undermined the confidence of large numbers of well-intentioned people in the judgment and the intentions of the responsible governmental authorities, because the facts have, after all, leaked out or have been suspected by the public, and they wonder why nothing has been said about it.

As I said before the National Academy of Sciences 2 years ago:

> So many of the public are already aware of the genetic damage produced by radiation that their morale is weakened and their apprehensions are increased when they see that the damage is denied by prominent sponsors of our national defense. Thus the door is opened for their acceptance of the defeatist propaganda which alleges that even the tests are seriously undermining the biological integrity of mankind. In this situation the only defensible or effective course for our democratic society is to recognize the truth, to admit the damage, and to base our case for continuance of the tests on a weighing of the alternative consequences.

Now when we do this, we conclude that the number of lives that will be seriously curtailed or injured throughout the world in future generations, as a result of the tests already held—supposing that they continue at this rate for perhaps 10 years longer—is in all probability in the hundreds of thousands or millions, and is therefore enormous. We should recognize that.

Despite all the uncertainties in regard to the exact figures, I think it was not possible to make clear to you how much careful work these estimates were based on, and the fact that although there may be an error of what we call a factor of 2 or 3, that is, that the true figures may be 3 times as much or only a third as much, nevertheless it is very unlikely that they should be less than, I would say, a third as much. In other words, the values given to you by Dr. Crow are those which are most likely in the light of present knowledge.

So I think we must recognize that the number of lives that would be seriously curtailed or injured will be in the hundreds of thousands or millions, and is therefore, enormous.

Senator ANDERSON. Doctor, you say that a number of lives seriously curtailed or injured from tests already held?

Dr. MULLER. Yes. I should modify that. I should say from tests held at the rate at which they have been held.

Senator ANDERSON. As they are now going?

Dr. MULLER. Yes.

Senator ANDERSON. That does make some difference.

Dr. MULLER. Yes.

Senator ANDERSON. That would damage, you think, hundreds of thousands or perhaps millions, and you say that is within a probability factor of 3?

Dr. MULLER. Yes.

Senator ANDERSON. A third as much or three times as much?

Dr. MULLER. I would put it at that.

Senator ANDERSON. Then it would be a very substantial number of lives if it is on the smaller side and an enormous number of lives on the larger side.

Dr. MULLER. Yes.

Senator ANDERSON. Doctor, do you believe that a number of geneticists agree with you in that point of view?

Dr. MULLER. I do, yes. They might differ as to where to put the factor. Some might say 2. Some might say 5 or 6.

Senator ANDERSON. This would sort of imply that there is no threshold.

Dr. MULLER. Yes.

Senator ANDERSON. That it starts immediately.

Dr. MULLER. Yes. That is because of the mechanism that Dr. Pollard explained. The thing strikes or it doesn't strike. If it strikes it does the damage even if there was only that one strike. He made the comparison with a lightning strike occurring in a large space and a long time, in which however that strike would be just as effective.

Senator ANDERSON. I ask that question because of the discussion we had this morning of having some group to come in and discuss these figures with a responsible group such as the Commission itself. You do feel that the geneticists could make a strong case in support of that?

Dr. MULLER. Yes, especially those who have worked in the field.

Senator HICKENLOOPER. Dr. Muller, in your prepared statement I notice you said with respect to that, and I quote from the prepared statement as follows:

> As I stated before the National Academy of Sciences 2 years ago, it has caused—

that is, certain conceptions or misconceptions—

> people to lend too ready an ear to the alarmists who declare that the genetic material of the human race is seriously endangered by the fallout from the test explosions themselves.

I do not know whether you got to that or whether you intended to do it or not.

Dr. MULLER. No. I read a more detailed statement on the subject, taken from the discussion that I gave before the National Academy

of Sciences. I read a part of that discussion. But I also stand by the statement as I have it in the text that you have just read.

Senator HICKENLOOPER. With respect to these numbers which you refer to as hundreds of thousands or millions, do I understand the connotation of that to be that that in fact is extremely minute as compared to all the human beings for ensuing years?

Dr. MULLER. Yes.

Senator HICKENLOOPER. Although it is large in numerical value standing by itself.

Dr. MULLER. That comes in the next sentence, you will see, where it says:

Nevertheless these injuries, being scattered over the whole earth and through hundreds of years—

I should have said thousands—

are relatively very few, in comparison with those due to other causes, including natural mutations. Moreover, the suffering to be entailed, although enormous in absolute terms, must be very small relatively to that which might follow from any serious mistake in the conduct of international relations.

Senator HICKENLOOPER. Therefore, Doctor, it becomes a matter of relative value in this particular field.

Dr. MULLER. Yes.

Senator HICKENLOOPER. Some things we may have to do, such as getting into war where we do not want to kill people, whether by bullet or disease or anything else, but we have to balance security and necessity against the hazard and strike a balance as to our conduct.

Dr. MULLER. Yes. I do not mean that when we strike that balance—I am not trying here to say which way the balance will be struck.

Senator HICKENLOOPER. I understand. You are making the point that someone must exercise judgment and determination in the light of all the circumstances.

Dr. MULLER. Yes. I would agree with Doctor Sturtevant that one life is a serious matter.

Senator HICKENLOOPER. Yes, without doubt.

Representative HOLIFIELD. You are really making a plea that all the facts be known before the decisions on policy are made.

Dr. MULLER. Yes.

Representative HOLIFIELD. And no facts be repressed.

Dr. MULLER. Yes.

On the other hand, the consequences of a full-fledged war, with its heavy irradiation of large numbers of people on both sides, would be inordinately more serious in its effects on the human genetic heritage as well as in its more direct effects. It is this consideration which, in my opinion, makes a continuation of test explosions a monstrous mistake of policy for both sides. Of course it would be absurd to expect one side to stop without the other. But a continuance by both sides would tend to lead the world nearer to a war that even with present techniques would result in the cataclysmic ruination of humanity in general.

May I add that the means of destruction are now so advanced on both sides that further advances by one side alone could not save it in the case of a war from becoming itself destroyed. By war I mean an atomic war, because I do not think you can have a world war any-

more without a thermonuclear or atomic war. I do not think it is realistic to suppose that you can.

Senator HICKENLOOPER. This illustration, I realize, is not exactly on all fours with the situation we have here today, but we are concerned with the danger and propriety of continued tests in the world.

Dr. MULLER. Yes.

Senator HICKENLOOPER. I presume all of us would earnestly hope that we never had to test atomic weapons. That perhaps would be the ideal. There are certain political factors that enter into those decisions, but by the same token I presume that we want to save thousands of lives in this country every year and we could just abolish the manufacture of automobiles and go back to riding horses. It seems to have struck a balance in the minds of people that transportation is important and we keep making automobiles, people keep getting killed by the thousands on the highways every year. We are all sad about that.

The point I was attempting to understand in my own mind is that there is a balance which someone must determine as to the ultimate good to either us or the world in this atomic field and whether or not we continue.

Dr. MULLER. I would accept that, except that I would rather say, "which every one has to help determine."

Senator HICKENLOOPER. Yes, indeed.

Dr. MULLER. I might add in this connection that in my talk to the National Academy 2 years ago I stated it to be my belief at that time that a continuation of the nuclear tests was necessary. I still think that this was the case at that time but I think the situation has changed since then. But there I am not speaking as a geneticist.

Senator HICKENLOOPER. I do not know whether I understand or not, but may I ask you, are you advocating that the United States stop testing weapons unless we get reliable agreement that other nations in the world would stop also?

Dr. MULLER. Of course not; no. The more that we can get people of the world to recognize the terrific damage that nuclear war will bring to them all, I think the more they will see the light on that point.

Senator BRICKER. Are you going to discuss later, Doctor, the answer to the question I asked this morning in regard to the experimentation that is going on in Russia?

Dr. MULLER. Yes. Just a little more here—I am on the last page on a somewhat different topic, but it is all related.

In order that the grave biological effects of radiation may receive due recognition and study—and I have tried to show you that they have not received due recognition and study—and may duly influence our policies and procedures, it is important that persons with a systematic background in genetics be placed in positions in which the decisions involving these matters are made. Truly this should be the case if there is any chance that genetic processes lie behind all the major damage done by radiation to man. Yet this is not the case at present.

For example—and let me not be misunderstood here—I have the highest regard for Dr. Shields Warren and for Dr. Brues and their associates, but I think it is important in this connection to point out that the official delegates of our country on the United Nations Scien-

tific Committee on the Effects of Atomic Radiation—and those are the gentlemen I have just mentioned—are neither of them geneticists. I am sure neither of them would wish to claim to be. I mean by that they would disclaim it.

Representative COLE. On that point, Doctor, do you know whether other members or official delegates from other countries on this committee are geneticists?

Dr. MULLER. There is one geneticist of bacteria, Dr. Appleyard of Canada. There is Dr. Caspersson from Sweden who studies chromosomes and other cell materials through the microscope. He is not exactly a geneticist. There is an alternate, Dr. Gopal Ayengar, from India who is a geneticist. Dr. Bacq of Belgium is in fields related to genetics.

So far as I know, those are the only ones that come near the subject of genetics who are on that committee. I stand to be corrected. They can, of course, and have, at least at the last meeting, I understand, had some geneticists present as consultants. Yet the chief discussions of that committee to date, so I have been told, have been on directly genetic matters, on these very questions here, especially on the effects on future generations.

You may remember that this country insisted that the delegates be chosen not by sciences but by countries and that the delegates by countries be chosen by the government and not by the scientific bodies. Some other countries put up strong resistance against that, but finally accepted it.

It may be noticed that most of the nongeneticists who deal with these matters, as I think is clear from the discussions of yesterday, are on the same side. They are on the other side from geneticists in regard to the major question of whether there is a threshold or whether the major effects on the body are linearly proportional to the dose all the way down to zero. That is an important issue in assessing the effects not only of the tests, but also of the peacetime uses of atomic energy.

I might say that when it comes to another body that is very important in this connection, the National Committee on Radiation Protection, we are in a better situation. Dr. Glass here, a very good geneticist, if I may say so, is a member of that committee, who was newly appointed just a few days ago. I was a member of one of the subcommittees for some years, although I am not sure whether I still am or not.

Representative COLE. Mr. Chairman, may I clarify the record? I understand Dr. Glass had been a member of this committee for 3 years.

Dr. MULLER. No, that is another committee. That is the Atomic Energy Advisory Committee on Biology and Medicine. I am now speaking of the National Committee on Radiation Protection which is under the auspices of the National Bureau of Standards and which is the one promulgating the permissible dose which we had explained to us the other day on the blackboard.

The latter committee do have, as I have said, a geneticist here and there. However, they have official representation from about 15 different organizations, mostly of a medical or governmental nature. Yet they do not have one official representative from any of the professional genetic organizations, such as the Genetics Society of America,

the American Society of Human Genetics, the American Genetic Association, or the Society for the Study of Evolution, all of which are in my opinion as closely concerned with this matter as for example the Radiological Society. Consequently, there is not sufficient representation among them of that genetic point of view of the mechanism which leads us to expect no threshold and to take the matter more seriously at small doses. This circumstance, I think, provides the reason why the record of this committee's decisions on the permissible dose, which as Dr. Taylor presented the matter yesterday appeared to show that they were so cautious, in actuality showed that the first dose they set was far too high, so that they had to set it lower. Then they found the second limit also was far too high and again they came down. And recently they found that the third limit in turn was too high and they came down once more. This does not indicate that they have been so cautious. It means they have not been cautious enough. The geneticists would not have set so high a permissible dose in the first place, on the basis of what we knew 30 years ago.

The grounds for the reduction in permissible dose that was made by the committee a few years ago, prior to the issuance of the National Academy's report, did not lie in considerations of genetic damage. For the permissible dose handbook specifically stated that this dose (of 0.3 roentgens per week) was set without regard to genetic effects. The geneticist members objected to that but it was carried anyway. In other words, it was known that the dose was considered too high on genetic grounds but it was adopted in spite of this, although it was acknowledged that it might be reduced again later.

Now that the Academy has made its report it has in fact been reduced a great deal more. I agree however with Dr. Glass, that it is probably due for even further reduction.

A similar attitude is reflected in the omission of any mention of the genetic effects of radiation in the courses on radiation in relation to health that are given both for our own people and for foreign selectees under AEC auspices at Oak Ridge. It is not enough to have biologists of some sort, or medical men, to reach decisions on these matters, unless they include a strong contingent of geneticists and of those who have a genetic point of view. Others are not likely to admit the danger from small doses.

In view of this situation and of the notorious resistance to the acceptance of genetic principles on the part of so many, not only of governmental appointees in the policymaking positions, but also of so many of the medical profession, a resistance that has prevented the medical profession for 30 years from duly protecting themselves, their technicians, and their patients when X-rays are used medically, and that has thereby subjected the reproductive cells of our population to very much more radiation than that from fallout—it is my opinion highly important that a National Radiation Health Institute be established as a part of the United States National Institutes of Health, but only if it contains a solid core of competent and versatile geneticists as one of its major features.

It is true that there is excellent research on the genetic and other effects of radiation being carried out in our country and that a considerable amount of it is made possible by the support or is done under the auspices of the AEC. This research, however, does not sufficiently

tion to public health and well-being, and the promotion of adequate measures in application of the conclusions reached.

If anyone wishes to ask some specific questions on the Russian aspect of the situation, I would be glad to take them up.

Senator BRICKER. I just wanted him to discuss the situation and whether they are conscious of the conclusions you have come to and are presenting to us and what research is being done there with regard to the effects of fallout.

Dr. MULLER. I have to infer from my knowledge of Russia derived from various sources, including firsthand information gained 20 years ago, that they are and will be having to follow our lead. We can't look to them for useful information at present along these lines. For, as I think most people realize, there was a purge of geneticists and an expurgation of the subject of genetics from teaching in the school and universities, from the boards of publication of journals, and from research institutes. It has not been taught to students for about 20 years. Most of the leading geneticists were somehow done to death, and I say this advisedly.

Chairman DURHAM. You say they will have to follow us. Do you think they would follow us?

Dr. MULLER. Yes.

Chairman DURHAM. Do you think so?

Dr. MULLER. Yes. There are a few geneticists left, of course. I think that some of the politicians in leading positions, since Stalin died, realize the folly of their old ways in regard to the subject of genetics. We have solid evidence that it is now possible to advocate the principles of genetics and to do some research in it.

A few of the old research workers are left, and there are said to be plans to give them positions in which they can resume their genetic investigations. However, the quacks have not by any means been disestablished yet, although they do not hold as commanding positions as they had before. One of the older geneticists, Dubinin (he was not old 20 years ago), was even rumored to have been selected as one of their delegates on the U. N. Scientific Committee on the Effects of Atomic Radiation. If so, Russia did better than most of the other countries in regard to that committee because Dubinin was a real "honest-to-goodness" geneticist.

Chairman DURHAM. You do think that Government officials are getting advice on genetics?

Dr. MULLER. They are beginning to get advice on genetics again. But I also note that the quack group are still strong. We have proof of that in publications and in the fact that in the Conference on Genetics that was held in Japan last September the Russians sent, I believe, four delegates, and all of them belonged to this quack school. So there is a division on the matter in Russia now.

Chairman DURHAM. What do you think is the reason for the Russians not permitting the genetic scientists to take part in the United Nations?

Dr. MULLER. The Russian, Dubinin, is a geneticist. Most of the other countries didn't seem to realize that geneticists were needed, for this committee seemed to assume that physicians would know about the subject. However, you will find very few physicians in this country that have an education amounting to anything in genetics.

Chairman DURHAM. Did I misunderstand you? I thought you said they would not permit him to go to the United Nations panel.

Dr. MULLER. No. I said I had heard, but I cannot verify, that Dubinin was to go to that meeting as an official Russian delegate. Whether he went or not I do not know. Perhaps someone here knows. I would be interested to know if he did.

Representative HOLIFIELD. Dr. Muller, you have spoken in two instances in your presentation here of the prolonged official reluctance and the curious official silence. Is it not true that you were invited to give a paper at the Geneva Conference a couple of years ago?

Dr. MULLER. Yes.

Representative HOLIFIELD. The Geneva Conference on Atomic Energy?

Dr. MULLER. Yes.

Representative HOLIFIELD. Did you give it?

Dr. MULLER. It was printed in the proceedings.

Representative HOLIFIELD. That is not quite an answer to my question.

Dr. MULLER. No, I did not give it there. I was prevented from giving it.

Representative HOLIFIELD. Who prevented you from giving it?

Dr. MULLER. The story has some complications of detail, but the essential thing is that it was called off by higher echelons of the AEC. That was actually shortly after this article of mine had appeared in Science that I quoted from earlier, in which I said there had not been enough airing of the matter.

Representative HOLIFIELD. Were you given any reasons as to why you were not allowed to give your paper?

Dr. MULLER. Yes; there was not room for me. Also they were sorry they had to notify me so late because they had only just received word so late from the International Committee. It was afterward found that it was not the International Conmmittee that had asked to have my paper excluded. They had approved of having it given. That expression was used, however, in the official letter written to me by the AEC authority.

Representative HOLIFIELD. Are there any further questions?

Representative COLE. Mr. Chairman, it had been my understanding that the committee was going to allow our staff specialists in this field to interrogate the witness in any area in which they felt there was some need for further amplification. I would like to inquire if Mr. Hollister might not have some questions of Dr. Muller. I would suggest that hereafter Mr. Hollister would be invited to interrogate. He is reluctant to inject himself into the interrogation.

Representative HOLIFIELD. The Chair has informed Dr. Tompkins and Mr. Hollister that they have the privilege of touching me on the shoulder and asking questions of any witness. They certainly do have that privilege. It was announced at the beginning of the meeting, and we certainly intend to allow them that privilege.

Mr. Hollister, would you like to ask some questions of Dr. Muller before he leaves the stand?

Representative PRICE. Before he does, is Dr. Muller's paper before the National Academy of Sciences included as part of the record?

Representative HOLIFIELD. Dr. Muller, Mr. Price's question was,

have you presented your paper that you were to give at the Geneva Conference as part of the record of your presentation?

Dr. MULLER. I had not intended to do so. I would like to present something more recent—two things in fact—one short paper, Potential Hazards of Radiation, which is now in press in the journal Excerpta Medica published in Amsterdam. It is six typewritten pages with some references.

Then a little statement that I gave out last October 20, 1956, that I think I could read. It is very short.

So devastating would be the damage done to both present and future generations by the nuclear explosions which any global war is likely to bring, that the great issue of today is not that of the relatively minor damage produced by mere tests of H-bombs, but that of taking all steps we safely can, such as the mutual discontinuance of these tests, which will tend to lessen international tensions and bring us nearer to all-round armament control. Unless this control is achieved in the short time open to us before thermonuclear weapons have become available to more countries still, and before intercontinental missiles have become a reality, we will find ourselves in a situation even more ungovernable and menacing than that of today.

I also wish to introduce a paper in the report by the World Health Organization study group on the effect of radiation on human genetics (see appendix, p. 1728). This report of the World Health Organization is being presented to the U. N. Committee on Radiation Damage and will be published next month sometime, it is expected. I have a copy of the paper of mine of that report here to include.

Representative HOLIFIELD. Thank you very much. Without objection they will be received.

(The document referred to, together with an article entitled "How Radiation Changes the Genetic Constitution" by Dr. Muller, follow:)

POTENTIAL HAZARDS OF RADIATION [1]

(By H. J. Muller, Indiana University)

Evidence has in recent years been accumulating for the broad conclusion that the great majority, if not all, of the damaging effects on life and health evoked by ionizing radiation are results of permanent changes produced in the genetic material: the chromosomes or their contained genes. These changes, when occurring in the genetic material of the germ cells, reach expression through reduction in the number of functional germ cells (infertility), increased mortality of zygotes of the first and subsequent generations (dominant lethals and detrimentals), and, in general, the alteration of one or more hereditary characteristics, that thereafter are transmitted in their new form (mutations).

Our knowledge of the nature of these effects, and of their manner of production, has been derived in the first place from studies of the descendants of exposed individuals, checked by cytological observations of cells derived from exposed germ cells. However, there is increasing ground for the inference that ionizing radiation produces changes in the genetic material of the somatic cells like those in the germ cells, and that it is these chromosomal and gene changes in the somatic cells that form the basis of most of the damage to the exposed individual himself, such as erythema and the various other aspects of radiation sickness, shortening of the life span, diverse malignancies, etc.

The primary changes in the genetic material, as disclosed by cytogenetic investigations on widely different forms of life ranging from viruses and bacteria to fungi, flowering plants, insects and mammals, may for convenience be divided into two groups. These are the *chromosome breaks*, that result in fragments the broken ends of which tend to unite with one another either in the old or in new arrangements, and the *point mutations*, that involve alterations at very localized positions on the chromosomes and are inherited according to Mendelian principles. It would take us too far afield here to discuss the proposi-

[1] Slightly modified version of article in press in June 1957 issue of Excerpts Medica (Amsterdam).

tions that there may be a fundamental similarity between the changes of these two groups, that they may integrade with and even overlap one another, and that, at any rate, the classification into one or the other group is in many cases uncertain. However that may be, the distinction remains of practical importance.

It is the point mutations which, although the more elusive of the two types of genetic changes, do the most harm in the long run. Even though evolution has come about by the natural multiplication of the very infrequent advantageous point mutations, the vast majority of them, whether arising naturally or induced by radiation, are of a detrimental nature, as is only to be expected of "blind" changes. Yet, contrary to popular opinion, mutations giving rise to conspicuous abnormalities, monstrosities, or freaks, are a great rarity, even after heavy doses of radiation. The point mutations induced in animal material by ionizing radiation have been found to be similar in their range of types and in the relative frequencies of those having different kinds of visible expression ("phenotype") to the naturally arising mutations, although the distribution of relative frequencies from gene to gene may be somewhat different for the two groups.

In considering the expression of a mutant gene we must distinguish between that which it has when homozygous, i. e. when inherited from both parents alike, and when heterozygous, i. e. from but one parent (the other having supplied a normal gene). Many mutant genes (possibly as many as one-fifth, as indicated by work on fruit flies) have such drastic effects when homozygous as to unconditionally kill the individual prior to maturity: these are the "lethals." The great majority of the remainder, when homozygous, cause some degree of impairment, even though relatively few of them give rise to readily visible abnormalities. In heterozygous condition their expression is usually much less pronounced than in homozygous condition and is very seldom recognizable; hence most mutant genes are termed "recessive." Yet even when heterozygous there is usually some slight, statistically important impairment of the capacity to live and reproduce. Hence, since the mutant gene is regularly transmitted to subsequent generations, it nearly always results, eventually, in the extinction (genetic death) of the line of descent carrying it, and this usually happens before the gene has had an opportunity to become homozygous.

These "genetic deaths" are seldom identifiable as such because the heterozygous individuals that suffer them show so little recognizable impairment. They represent the price paid by any population in preventing an unlimited accumulation of mutant genes within it. Studies on the frequencies of natural mutations in Drosophila, the mouse, and man, and of the effects of inbreeding in man, agree in indicating that each person carries on the average, mainly in heterozygous condition, at least four times as many mutant genes as would have been enough to kill him outright if they had been homozygous. It is further indicated that, in the mainly heterozygous condition in which the mutant genes actually occur, they tend, by their cumulative action, to cause the genetic death of at least 1 person in 5.

When the mutation rate is raised by exposure to radiation the frequency of genetic death is correspondingly raised, over a number of generations that is inversely proportional to the degree to which the mutational damage is expressed in the individual's of any given generation. Russell's studies on the mutations of 7 genes in mice show that some 30 r. delivered to the immature germ cells constitutes the "doubling dose," in that it induces in them as many mutations as arise naturally per generation. It is unlikely that in man the doubling dose is more than twice as high, and it may even be somewhat lower than in the mouse. At any rate, the correctness for man of this order of magnitude is indicated by studies of Turpin and Lejeune and of Macht and Lawrence, and is not contradicted by the lack of statistically significant findings in the studies made in Japan by Neel and Schull.

Since there is much evidence indicating a linear relation between the radiation dose and the frequency of the induced point mutations, even at extremely low doses, and the exactly cumulative nature of these radiation effects, it becomes possible to arrive at probable estimates of the minimum damage done to subsequent generations by any given chronic or acute exposure of parents. In view of the dearth of conspicuous abnormalities, one of the best overall measures of

this damage lies in a measurement of mortality, expressed for instance as length of life. The criterion has recently been used by Russell in his demonstration that neutrons applied to male mice cause a shortening of the average life-span of their progency that is about as great as that caused in the directly exposed individuals: nearly 0.1 percent of the life-span per rep. Since, however, the effects on the progeny are likely to reappear throughout scores of generations before being terminated by means of genetic deaths, the total damage to the descendants is many times greater than to the exposed individuals.

Chromosome breaks, like point mutations, are induced with a frequency directly proportional to the total dose of radiation, regardless of how concentrated or dispersed the treatment has been. Some of the rearrangements of chromosome parts result from single breaks, and some from two or more breaks lying in the course of the same ionizing particle. The frequency of either of these types of rearrangements, like that of the individual breaks, varies directly as the total dose, regardless of its distribution in time. Other rearrangements, that result from a combination of fragments derived from independent breaks occurring within the same few minutes, have in consequence of this mode of origin a frequency that is insignificant when the radiation has been of low intensity but that rises rapidly with the intensity, approximately as its square, and that becomes of major importance at high dose rates. Thus chronic or repeated low exposures give rearrangements that are linearly related in frequency to the total accumulated dose, while higher dose-rates give disproportionately numerous effects.

A cell in which one or more chromosomes have been structurally changed by breakage will continue to function normally until cell division occurs. At that time the altered chromosomes often give rise to chromatin bridges that connect the daughter nuclei, interfere with their further multiplication, and ultimately result in death of the affected cell line. In the absence of a bridge, the daughter or descendant cells may come to lack parts of chromosomes and/or to have other parts in excess, and the resulting unbalance of gene proportions ("aneuploidy") tends to impair and even kill the descendant cells. Happening in the germinal line, these phenomena are expressed as infertility of the exposed individuals, and as lethality among embryos of later generations.

The chromosome damage, leading to postmitotic cell impairment and death, is also induced in somatic (body) cells. It provides an interpretation of such phenomena as the "law of Bergonie and Tribondeau" (relating the degree of tissue damage to multiplicational activity), the radiation death of individual somatic cells in tissue cultures (as in work of Puck and Marcus), and the delayed production of minute cataracts in irradiated lenses of the eyes. That it is the chromosomes rather than the protoplasm of cells which are ordinarily the seat of the more significant radiation changes, leading to cell death, has been shown in numerous studies, among them those of A. R. Whiting, Zirkle and Bloom, and Ulrich. It is only when a given number of cells has been destroyed within a given space and time that certain visible symptoms appear, such as reddening of the skin, reduction in number of white blood cells, intestinal hemorrhage, etc. However, there is no threshold for the individual cell effects, and analyses such as that of Quastler are increasingly implicating them as the basis of the clinical manifestations.

It is almost certainly through the individual cell deaths and impairments that minute doses of radiation, long continued or repeated, exert their action in shortening the life-span of the exposed individual. This effect, first analyzed by Boche and then by Sacher, has been calculated to cause a reduction in length of life of the order of several days for every roentgen unit received by the body as a whole during a person's lifetime.

On the other hand, leukemia and some other malignancies, the induction of which may also be linearly dependent upon radiation dose, are considered by geneticists as being more probably results of point mutations in somatic cells than of chromosome breaks. From the conclusions of Lewis it may be calculated that for a population of 160,000,000 with a lifespan like that in the United States each absorbed roentgen of whole-body radiation would result in some 10,000 cases of leukemia during their lifetime, while one-tenth the "maximum permissible dose" of strontium 90 would result in some 55,000 cases.

The present population of the United States has been reckoned to receive, on the average, some 5 r. of radiation to the gonads from medical exposures alone before the age of 30 (see Laughlin and Pullman) but the amount from all diagnoses and treatments may well be double this (see Schubert and Lapp). About 3 r. are received from the natural background radiation. The amount

from atomic test fallout is as yet much less, and is said to be of the order of .1 r., although atomic warfare, or insufficient precautions in the peacetime use of atomic energy, could raise it enormously. The present exposure of Western populations, caused largely by fluoroscopy and by roentgenograms of the lower trunk, is not enough to cause concern in regard to shortening of life of the exposed generation, but its effect on future generations must be a good deal greater. The same consideration applies to occupational exposures. It has led to the recent recommendations for intensification of radiation precautions promulgated by the National and International Committees on Radiation Protection, and the committees on the genetic effects of radiation of the National Academy of Sciences (United States), the Medical Research Council (Great Britain), the World Health Organization, and the United Nations. It has recently been reflected in increasing activity in this direction in medical and dental circles.

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[Reprinted from the Bulletin of the Atomic Scientists, November 1955]

How Radiation Changes the Genetic Constitution

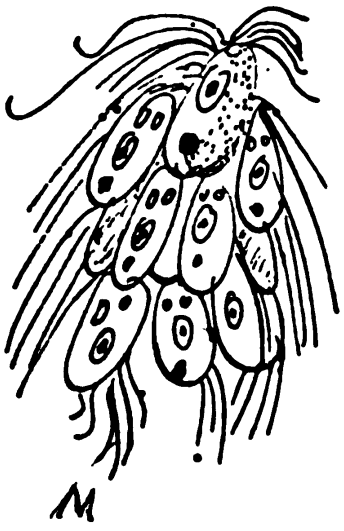
H. J. Muller

Dr. Muller, professor of genetics at Indiana University, prepared the following paper for presentation at the United Nations Conference on the Peacetime Uses of Atomic Energy, at Geneva. Although the paper was not given orally, it will appear in the published proceedings of the Conference.

The changes in the genetic constitution produced by ionizing radiation may for convenience be classified into two major groups: *chromosome aberrations* and *point mutations*.

The chromosome aberrations consist of losses and additions of whole chromosomes or chromosome parts and/or alterations, called structural changes, in the alinement of chromosome parts. Structural changes are caused by the breakage of one or more chromosomes at two or more points, followed by the junction of the fragments at their broken ends, so as to form a new arrangement; that is, a new linear sequence of their component hereditary particles or genes.[1]

Point mutations are changes confined to regions of the chromosomes so small that no loss or addition or change in arrangement of genes can be demonstrated by microscopic examinations or breeding tests. Since structural changes range from "gross" to those so minute as to be at the limit of being detectable as such, there are doubtless other cases of substantially the same kind, but below that limit of size, which become included among the point mutations. However, there is reason to infer that many of the point mutations produced in animals by radiation are not of this kind, but involve changes within the individual genes, and are therefore to be considered as "gene mutations." By this it is meant that these changes are restricted to genetic elements too small to be divided either by the process of normal hereditary recombination (crossing over) or by that of gross structural change. This seems to be true also of the great majority of genetic differences that exist naturally between individuals of the same species; that is, they appear to have arisen as gene mutations.



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[1] H. J. Muller, J. Genet, 40: 1–66 (1940).

CHROMOSOME ABERRATIONS

The chromosome aberrations produced by radiation in the cells of somatic tissues that replenish themselves by proliferation cause necrosis in much of the tissue descended from these cells and abnormality in much of the surviving descendant tissue. This constitutes a major source of delayed radiation damage, some of it never repaired, in the exposed individual himself. The same series of events, occurring among the immature germ cells of the exposed individual, can result in his or her partial or complete sterility. Among mature and nearly mature germ cells, especially spermatozoa, there is a much higher incidence of induction of these chromosome changes, for any given dose of radiation, than among immature germ cells or somatic cells.[1] Recent evidence[2] confirms the inference[1] that this peculiarity depends upon the chromosomes being in a condensed (tightly spiralized) condition and that it therefore applies also to cells that are in mitosis at the time of irradiation.

Mature sperm or eggs in which chromosome aberrations (actual or potential) have been induced, function in fertilization, but many of the resulting embryos die in consequence of their abnormal chromosome content. Other embryos, in which there has been gross structural change without excess or deficiency of chromosome parts, develop into normal adult individuals. However, when these seeming normals reproduce, recombination occurs between the structurally changed chromosomes derived from one parent and their normal homologues derived from the other parent. In consequence of the nonmatching linear arrangement of the genes from the two parents, about 50 percent of the germ cells now produced have excesses and/or deficiences of chromosome content. These germ cells usually function, but give rise to embryos (of the second generation after exposure) which die in utero at an early stage.[4] This mortality of embryos tends to be repeated over an indefinitely long series of generations. For half of the surviving embryos of such a line of descent, although not themselves containing the lethal excesses or deficiencies, have the grossly changed linear arrangement of genes that, by recombination, again gives rise to these effects.

In modern human populations, there is a tendency to compensate or even overcompensate for reductions in the frequency of viable births, by purposely increasing the number of pregnancies.[5] Hence damage of this kind, once induced, does not tend to die out rapidly but may even spread.

Fortunately there are several factors which serve to limit the frequency with which these cases of inherited abortions are produced. One is the fact that the period spent by male germ cells in a mature or nearly mature state averages, at the very most, a few months, whereas they usually spend some 25 years or more—well over 100 times as long—as immature germ cells, relatively insusceptible to the induction of chromosome aberrations. Although the relative lengths of the corresponding periods for female germ cells are not well established, the germ cells are, even when nearly mature, much less susceptible than spermatozoa to the induction of the gross aberrations that cause inherited abortion.[6] It may be concluded that more than 99 percent of the germ cells which function after a given exposure of limited duration (comprising only a few days or weeks) were at the time of that exposure in an immature stage, relatively insusceptible to the induction of chromosome aberrations. In them, aberrations of all kinds were induced with far lower frequency than point mutations.

Even in the less than 1 percent of germ cells that are exposed to radiation of beta or gamma type during their susceptible stage, gross structural changes of chromosomes will be produced at a low frequency, relatively to point mutations, unless the total dose of radiation received in that period is fairly high, of the order of a hundred or more r. (roentgen) units. This is because the

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[1] See footnote, p. 1069.

[2] However, some immature germ cell stages are much more susceptible to chromosome alteration than they appear to be when judged by the frequency with which such alterations are found later, on analysis of offspring derived from the cells that had been exposed while immature. This is because the descendant cells derived from those immature germ cells in which the chromosomes had been altered so often die out, and have their places taken by compensatory multiplication of descendant cells derived from those immature germ cells in which the chromosomes had not been altered. This consideration does not apply in the case of point mutations. (Note added October 5, 1955.)

[3] I. I. Oster, Excerpta Medica (8th Internat. Cong. for Cell Biol. No. 8: 406 (1954).

[4] P. Hertwig, Z. indukt. Abstammungs-u. Vererbungslehre 79: 1–27 (1940). P. C. Koller, Genetics 29: 247–63 (1944). G. D. Snell, Amer. Naturalist 68: 178 (1934).

[5] Amer. J. of Human Genetics 2: 269–78 (1950).

[6] Roentgenol. Radium Therapy 41: 248–55 (1939).

production of these aberrations requires at least two chromosome breaks, and these are usually produced independently, by the tracks of different fast particles. On account of being in this sense double or multiple events, these aberrations vary in their frequency according to an exponent of the dose of radiation higher than one (commonly, about 1.5).[1] On the other hand, the point mutations vary as single events, according to the dose itself. Thus, as the dose is diminished, they do not drop off as fast as the structural changes do, and the latter become rare, relatively to the former.

It follows from the above considerations that inherited abortion caused by structural change is a relatively insignificant danger even in the case of a large dose of beta or gamma radiation that has been received in small fractions of not more than a few r. per month. If the amount received in any month is higher than this, however, measures should be taken to avoid this damage. These measures would consist in the prevention or avoidance of conception until the passage of several months after the high exposure. With a very high dose, however, all but the first month of this period would be sterile anyway.

When the exposure has been to alpha or neutron radiation, the production of gross structural changes tends to vary with the dose itself instead of with a higher exponent.[7] This is because both the breaks participating in such an aberration are usually produced by activations arising from the track of the same fast atomic nucleus. In consequence, both of this proportionality of the frequency of structural change to dose with this type of radiation, and of the fact that the more densely crowded activations from such radiation are actually more efficient in breaking the chromosomes, much lower doses, in reps, of neutrons or alpha rays than of gamma, beta, or X-rays give significant numbers of structural changes. Hence, the rule of not reproducing within some months after exposure should be applied in this case of much lower doses when the radiation has been of these types. In order to gage how low this limit should be placed, it should be taken into consideration that even 5 reps of neutrons, applied to spermatozoa, may be estimated to induce inherited abortion, based on gross structural change in some 1 to 6 among every thousand viable individuals derived from these spermatozoa.[8]

The frequency of natural occurrence of gross structural changes giving inherited abortion has not been studied extensively in mammalian populations, but it is known to be low. The highest recorded figure[9] is about 6 percent. Among offspring from spermatozoa treated with 500 r. X-rays, 25 percent have been reported by 2 observers.[10]

CHARACTERISTICS OF NATURAL POINT MUTATIONS

Among the genetic changes induced by exposure to radiation from artificial sources the point mutations are far more frequent and significant that the chromosome aberrations. Among the genetic changes that arise from natural causes (those somewhat misleadingly referred to as "spontaneous") the point mutations are still more frequent and important, as compared with the chromosome aberrations. Any ordinary population contains a large accumulation, or "load," of these natural point mutations, which have arisen in the course of many past generations. If any new point mutations are induced by radiation these are added to this already existing load of mutations. They thereupon become lost to view among the latter, in the sense that, with rare exceptions, the origin of any individual point mutation cannot be traced to the radiation. Thus, in order that radiation mutations may be viewed in due perspective, certain salient facts about the natural mutations should first be passed in review.

Natural point mutations occur sporadically. They are not individually controllable. Any such mutation may be thought of as resulting from an accidental ultramicroscopic encounter between a gene and some atom group, particle, or photon to which the gene happens, under the circumstances, to be vulnerable. It is probable that, on occasion, instead of the original or "mother-gene" becoming

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[1] See footnote, p. 1069.

[7] A. Catsch, O. Peter, and P. Welt, Naturwissenschaften, 32: 230–31 (1944). N. H. Giles, Jr., Proc. Natl. Acad. Sci. U. S. 26: 567–75 (1940). N. H. Giles, Jr., Genetics 28: 398–418 (1943). H. J. Muller, Amer. Naturalist 88: 437–59 (1954). H. J. Muller and J. L. Valencia, Records Genet. Soc. Am. 20: 115–16 (1951); Genetics 36: 567–68 (1951).

[8] G. D. Snell, Proc. Natl. Acad. Sci. U. S. 25: 11–14 (1939).

[9] P. Hertwig, Biol. Zentr. 58: 273–301 (1938). P. Hertwig, Z. indukt. Abstammungs-u. Vererbungslehre 79: 1–27 (1940).

[10] W. L. Russell, "Genetic Effects of Radiation in Mammals," Radiation Biology (edit. by A. Hollaender), McGraw-Hill Co., New York, Vol. 1, Pt. II, Chapt. 12, pp. 825–59 (1954).

altered, the accident causes a misstep in the construction of the "daughter-gene," but the effect is much the same as if the old gene had itselm mutated. In either case, point-mutational changes are permanent. This implies that the changed gene tends to be very stable, as the original gene was, and that in reproducing it continues to give rise to daughter genes like itself, that is, in this case, of the new type. Thus it "copies itself" through an indefinite succession of generations.[11]

The frequency of mutations in general is influenced, however, by many conditions. Thus, cells in certain developmental stages have mutations occurring more frequently in them, in other stages less frequently.[12] There is some evidence that markedly detrimental disturbances in the cellular biochemistry, of whatever nature, tend to favor the occurrence of mutations, while the functioning of the cell within its normal range is associated with a low mutation frequency. Certain special substances, such as the mustard gas series, some organic peroxides and expoxides, and triazine are so conducive to mutation that they have been termed "mutagens." [13] Some of them can in fact be used to induce mutations at about as high a frequency as with radiation. When the distribution of relative frequencies of the different types of mutations induced by one mutagenic agent is compared with that induced by another, or with that of spontaneous mutations, considerable differences are often found, even though most types of mutations produced by one agent are also produced to some extent by any other, and also arise spontaneously but at a lower rate.[14]

The partial selectivity of action of mutagens does not give evidence of being of such a nature as to result in the mutations produced by a given agent, or under given conditions, being better adapted, as a group, for life in the presence of that agent, or under those conditions, than are the mutations which arise under other circumstances. That is, mutations arising independently of radiation like those produced by radiation are, so far as the organism is concerned, accidents, not adaptive responses. There is evidence indicating that the organism has, through a long period of evolution, been selected for the maintenance of biochemical operations which give it as low a frequency of "natural" mutations as can practicably be attained just as it has been also selected to react in such ways as to minimize the occurrence of other accidents.[15]

It is entirely in line with the accidental nature of natural mutations that extensive tests have agreed in showing the vast majority of them to be detrimental to the organism in its job of surviving and reproducing, just as changes accidentally introduced into any artificial mechanism are predominantly harmful to its useful operation.[11] According to the conception of evolution based on the studies of modern genetics, the whole organism has its basis in its genes. Of these there are thousands of different kinds, interacting with great nicety in the production and maintenance of the complicated organization of the given type of organism. Accordingly, by the mutation of one of these genes or another, in one way or another, any component structure or function, and in many cases combinations of these components, may become diversely altered. Yet in all except very rare cases the change will be disadvantageous, involving an impairment of function.

It is nevertheless to be inferred that all the superbly interadapted genes of any present-day organism arose through just this process of accidental natural mutation. This could take place only because of the Darwinian principle of natural selection, applying to the genes. That is, on the rare occasions when an accidental mutation did happen to effect an advantageous change, the resultant individual, just because it was aided by that mutation, tended to multiply

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[11] H. J. Muller, Second Internat. Cong. Eugenics, N. Y., Abstracts, p. 7–8 (1921). H. J. Muller, Genetics 3 : 442–99 (1918). H. J. Muller, J. Exp. Zool. 81 : 443–73 (1920). H. J. Muller, Amer. Naturalist 56 : 32–50 (1922). H. J. Muller, "Mutation," Eugenics, Genetics, and the Family, Williams and Wilkins, Baltimore, Vol. I, pp. 106–12 (1923). H. J. Muller, Genetics 13 : 279–357 (1928).

[12] H. J. Muller, Yrbk. Amer. Philos. Soc. for 1945 : 150–53 (1946). H. J. Muller, Genetics 31 : 225 (1946). H. J. Muller, unpublished data.

[13] C. Auerbach, Cold Spring Harbor Symposia Quant. Biol. 16 : 199–218 (1952). C. Auerbach and J. M. Robson, Nature 157 : 302 (1946). M. J. Bird, J. Genetics 50 : 480–85 (1952). M. Demerec, G. Bertani, and J. Flint, Amer. Naturalist 85 : 119–36 (1951). K. A. Jensen, I. Kirk, G. Kolmark, and M. Westergaard, Cold Spring Harbor Symposia Quant. Biol. 16 : 245–62 (1952). J. A. Rapoport, Compt. Rend. Acad. Sci. U. R. S. S. 54 : 65–67 (1946). J. A. Rapoport, Bull. Biol. Med. Exp. U. R. S. S. 23 : 198–201 (1946). J. A. Rapoport, Compt. Rend. Acad. Sci. U. R. S. S. 61 : 713–15 (1948) O. Wyss, J. B. Clark, F. Haas, and W. S. Stone, J. Bact. 56 : 51–57 (1948)

[14] M. Demerec, Proc. Amer. Philos. Soc. 98 : 318–22 (1954).

[15] H. J. Muller, Genetics 3 : 422–99 (1918). A. H. Sturtevant, Quart. Rev. Biol. 12 : 464–67 (1937).

more than the others. By the continuance and repetition of this process, the type that had been normal became supplanted by other types, that were at least better adapted for life in certain particular environments, in in certain ways. Thus, the mutant gene of the previous era became the normal gene of today, and the whole system of genes of the species tended to become even more differentiated and highly organized. Yet at each stage the great majority of new mutations, if examined before being put through the sieve of selection, must have been detrimental to life or to reproduction, as they are today in all species studied, no matter what the degree of advancement of the species.

As important for the survival of a species as the differential multiplication of the few better adapted mutants is the reduction in number and eventual dying out, in competition with the "normal" type, of the much more numerous mutants that are less fit than the normals. Since each generation supplies a fresh crop of these mutations, to be added to those inherited from earlier generations, it is obvious that without this negative selection the system of genes would undergo continued decay. Thus after a time it would become completely heterogeneous, disorganized, and degenerate.[18] In the past, only natural selection has saved it. This selection makes it practically inevitable that any detrimental mutation, no matter how small its harmful effect, will in the long run become limited by tipping the scales against some descendant who carries it, causing his premature death or failure to reproduce.

However, this dying out of the unfit mutants is in most cases rather long delayed. One reason for this delay is the fact that mutant genes are in the great majority of cases heterozygous, that is, present in individuals who have received the corresponding normal gene from their other parent, and that in such a situation the normal gene usually produces most of the effect. The normal gene is for this reason said to be "dominant," and the mutant gene "recessive," even though the mutant is seldom completely without expression when heterozygous.

Another reason for the delay in the dying out of mutant genes lies in the fact that even in those relatively rare individuals who are "homozygous" for a given mutant gene, by reason of having inherited that same gene from both parents, the amount of abnormality is often not very great. Hence, even in this situation the gene usually confers a much less than 100 percent risk of premature death, or of failure to reproduce. It may be noted in this connection that the idea that most mutations are monstrosities or freaks is a popular misconception. In fact, only a tiny minority of mutations cause very conspicuous visible abnormalities.

CALCULATION OF NATURAL MUTATIONS PRESENT IN A POPULATION

The total number of point mutations (or, more correctly, of point-mutant genetic conditions) present in any population at a given time is a product of two interacting numerical factors. The first factor, a, is the total number of new point mutations that arise in one generation. The second factor, b, to be multiplied by the first, is termed the persistence. It represents the total number of individuals of successive generations by whom, on the average, any given mutation, present at first in one individual, comes to be inherited.[19] This same relation holds for mutations of any particular type as well as for the totality of mutations.

Obviously b, the persistence, depends upon the ability of the individuals carrying the mutation to live and breed, as compared with normal individuals. If, for simplicity, we assume the whole population to be of stable size, then b, for the average mutation, or for any given type of mutation, is the reciprocal of c, the average chance that an individual who has inherited it will be killed prematurely, or will fail to reproduce, as a result of the one or more functional impairments occasioned in him by that mutation. In getting this average chance of elimination, c, we must estimate the relative frequencies of individuals heterozygous and homozygous for the mutation, and multiply the chance of elimination of each of these types, taken separately, by its relative frequency. When this is done it is found that usually, despite the much smaller

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[18] See footnote, p. 1071.

[19] H. J. Muller, Amer. J. Human Genet. 2: 111–76 (1950). H. J. Muller and S. L. Campbell, unpublished data. (This reference was omitted from original. Note added October 5, 1955).

detrimental effect in the heterozygous individuals, their relatively large numbers cause most of the eliminations, and most of the total genetic damage to the population, to occur in this group. Thus, in most cases, the homozygous group can for practical purposes be ignored.[16]

In order to apply this method of calculation to human populations we must first have estimates of *a* and *b*. At present such estimates are very indirect, and serve only to indicate a broad range, within which, somewhere, the actual value is probably located. The fruitfly *Drosophila* has thus far been the only organism in which anything like a direct approach has been made to an observed value for either *a* or *b*, and even here the results are subject to very large errors. In this material it can be estimated that, in a population of 100 million, *a*, the number of new mutations arising naturally per generation that becomes transmitted to the next generation, is on the average at least 8 million, and that *b*, the persistence or average number of individuals of successive generations which finally come to inherit any given mutation, is considerably more than 20 and probably more than 40. This makes *ab*, the number of mutations carried by the population of a hundred million in any given generation, probably more than 320 million, that is, probably more than three per individual.

The estimate of *a* for *Drosophila* was obtained by first taking the observed frequency, 0.18 percent, with which "recessive" fully lethal mutations (those that invariably kill homozygous individuals) usually arise in the X chromosome per germ cell per generation when no mutagenic treatment is used.[15] This figure was then multiplied by 6, the ratio of recessive lethals in all the chromosomes to those in the X chromosome. This figure had to be obtained from experiments in which radiation was applied to spermatozoa.[17] The product, 1.08 percent, representing all lethals, was in turn multiplied by 4, the ratio which all mutations detrimental enough to have been detected by a given technique were found to bear to fully lethal mutations. This figure 4 also was based on radiation mutations.[18] Finally, the second product, 4.3 percent, was multiplied by 2, because each individual results from 2 germ cells, and the resultant percent, 8.6, was multiplied by 100,000,000, the number assumed to exist in the population.

That the application of the ratio 6, derived from radiation work, to natural mutations is legitimate has been shown by special tests. However, among natural mutations as a group, the ratio of all mutations to lethals is probably a good deal higher than 4, the ratio found among mutations produced by irradiating spermatozoa. For the radiation mutations include a greater proportion of structural changes and these are more often lethal. This is one reason why the final figure for *a* is very conservative. The other reason is that the methods of detection used failed to find mutations that produced less than about 10 percent risk of premature death, even if they caused considerable infertility, and such mutations may have been relatively numerous.

The figure for *b* is based on tests, carried out independently by two groups of investigators,[18 19] to determine how much risk of premature death is conferred by a "recessive" lethal mutation when it is heterozygous. In both cases an average figure of about 8 percent to 5 percent risk of death was obtained. This would result in only 1 heterozygous individual among some 25 being killed and would hence allow the average lethal a persistence of 25. That is, it would tend to be passed on to some 25 individuals, on the average, before it died out. Since, however, a considerable majority of mutations are not so detrimental as to be fully lethal when homozygous, and most of them are probably not even 50 percent lethal when in that condition, the figure of 1 in 25 (4 percent) for the risk of death when heterozygous must be considerably higher than that holding for the average mutation, and the persistence, being the reciprocal of this, would be considerably higher than 25. That is why 40 was used as a better guess for *b*, but the observed distribution of mortalities indicates that even it is likely to be too low.

Before we can convert our figure *a* for newly arising mutations in *Drosophila* into a corresponding figure *a* for man we must obtain some indication of the

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[15] See footnote, p. 1072.
[16] See footnote, p. 1073.
[17] R. L. Berg, Genetics 22 : 225-40 ; 241-48 (1937).
[18] J. J. Kerkis, Summ. Commun. XV. Int. Physiol. Congr. 198-200 (1935). J. J. Kerkis, Izv. Akad. Nauk SSSR 75-96 (1938). H. J. Muller, Verh. 4. Int. Kongr. Radiol. 2 : 100-02 (1934). N. W. Timofeeff-Ressovsky, Strahlentherapie 51 : 658-63 (1934). N. W. Timofeeff-Ressovsky, Nachr. Ges. Wiss. Gottingen N. F. 1 : 163-80 (1935).
[19] C. Stern, G. Carson, M. Kinst, E. Novitski, and D. Uphoff, Genetics 37 : 418-49 (1952). C. Stern and E. Novitski, Science 108 : 538-39 (1948).

ratio of mutation frequency in *Drosophila* to that in man. As yet the only line of approach to this problem lies in a comparison of the frequencies, in the two species, of natural mutations that produce certain specific effects, and that may be inferred to occur at given highly limited positions in the chromosomes. Although the evidence of this kind is meager and imperfect, there is enough of it to show that in *Drosophila* a mutation of any one specific type, located in a specific chromosomal position (so as to give rise to what is technically known as an "allele" or "pseudo-allele" of some preexisting mutation) arises, on the average, with a natural frequency of between 1 in 100,000 and 1 in 300,000 germ cells, the most likely figure being about 1 in 200,000.[20] In mice there is little published data of this kind as yet but it would indicate a figure in the range between 1 in 40,000 and 1 in 400,000 or, most likely, about 1 in 140,000.[21] In man, an estimate of between 1 in 50,000 and 1 in 100,000 has been arrived at, on the basis of a much larger amount of data than in either mice or *Drosophila*, but the uncertainties of the methods used in man are much greater.[22] These apparent differences in mutation frequency between the three species may well correspond to the different numbers of cell divisions which take place in their respective reproductive cycles, since these numbers for flies, mice, and men are related about as 1 : 1.5 : 2. At any rate, it is likely that the average frequency of mutations of any specific type in man is higher than in *Drosophila*, probably from 2 to 4 times as high. To be conservative, we will adopt the lower figure, 2.

It is, however, likely that the ratio of frequencies of specific mutations in man to those in the fly would not be nearly as high as the ratio of total mutation frequencies in man to those in the fly. For man, and mammals in general, give evidence of having a more complicated organization, all told, than the fly, especially when the complications of the nervous system are taken into account. Mammals may therefore be expected to have a more complex germ plasm than flies, one in which a larger number of different kinds of mutations of specific types can occur. This agrees with existence of a larger amount of the genetic substance, polymerized deoxyribonucleic acid, in mammalian than in fly chromosome sets. Therefore we are in all probability obtaining a low minimal figure for a in man if we multiply the *Drosophila* a by only two.

For the value of b in man or other mammals there is as yet little basis for a decision. The existing indications point strongly to the conclusion that natural mutations in mammals in general, including man, are, as in *Drosophila*, prevailingly recessive, yet not completely so. Moreover, they certainly include a fairly abundant group of "recessive" lethals, but it is probable that mutations having a lesser degree of detriment are more frequent than lethals. At this preliminary stage of our knowledge of the subject, then, we have little ground for using a markedly different value of b for mammals than for *Drosophila*.

The figure of about 6.5 is thereby arrived at as a minimal one for the content of recessive, definitely detrimental mutations (including lethals) per individual human being. In a preliminary calculation using related methods, the figure 8 was arrived at.[23] These estimates, as recently shown by Slatis,[24] can be checked in a more direct way. The method consists in observations of the frequency with which homozygous individuals, showing the more definite abnormality often associated with a homozygous mutation, appear among the offspring of marriages between close relatives. Application of this technique has led Slatis, very tentatively as yet, to the figure 8 as the most probable present approximation to the number of natural mutations of the kind in question for which a person is, on the average, heterozygous. This method now needs to be applied on a much larger scale but the present result is enough to be reassuring, in indicating that our mode of calculation is giving figures of the right order of magnitude.

It should be emphasized that in these calculations we are dealing only with these mutations which are detrimental enough to give a "sizable" risk of genetic extinction by way of premature death; that is, one as great as about 0.5 percent in the case of the heterozygous individual, or 10 percent in the case of the homozygous one. We do not know how many mutations arise which are less harmful than this, or which cause extinction mainly by their interference

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[20] See footnote, p. 1073.
[21] H. J. Muller, J. L. Valencia, and R. M. Valencia, Rec. Genet. Soc. Amer. 18 : 105–06; and Genetics 35 : 125–26 (1950).
[22] W. L. Russell, Cold Spring Harbor Symposia Quant. Biol. 16 : 327–35 (1952).
[23] J. B. S. Haldane, Proc. 8th Internat. Cong. Genet. (1948); Hereditas 35 (Suppl.) : 266–78 (1949). J. V. Neel, and H. F. Falls, Science 114 : 419–22 ((1951).
[24] H. M. Slatis, Amer. J. of Human Genet. 6 : 412–18 (1954).

with reproduction, not with life itself. However, even if there are relatively few, those few which have an average grade of detriment within the same order of magnitude as the frequency of their origination by mutation, will accumulate so as to be inordinately numerous in the population. They will provide a very considerable proportion of the superficially observable genetic variability. Moreover, the frequency of the different types of mutations of this group will differ greatly from region to region, in response to differences in the conditions of selection, as well as to random influences.

Since the frequency with which mutant genes of any given degree of detrimental effect exist in the population at any one time is the product ab, where b is inversely proportional to c, the degree of detrimental effect, it is evident that the existing mutant genes have a distribution, with respect to their harmfulness, very different from the distribution to be found on examining mutations as they arise. For, among the mutant genes as they exist in the population as compared with them at their origination, the less harmful ones are (in inverse proportion to their harmful effect) more numerous than the more harmful ones. For that very reason each slightly harmful mutation that arises tends to cause as much detriment to the population as a whole in the end as each drastically harmful or lethal mutation does, since it compensates for its relatively small degree of harm by afflicting correspondingly more individuals. In consequence, the total amount of genetic damage done to a population by mutations is much more closely proportionate to the total frequency of mutations arising per generation (a/N, where N is the number of individuals in the population) than to the frequency of mutations existing in the population (ab/N).[24] If a is raised or lowered, however, it may take scores of generations before its changed value becomes proportionately reflected in the altered average fitness or mutational load of the population. A similar lag occurs if b is altered, as happens when the rigor of selection is increased or decreased.

CHARACTERISTICS OF POINT MUTATIONS PRODUCED BY RADIATION

In the plant material studied by Stadler,[25] evidence was obtained, based on the intensive study of a few types of mutations, that the great majority of apparent point mutations induced by radiation probably consisted of losses of a small section of a chromosome including more than one gene, unlike what was usually true of the natural mutations. In the animal material best studied with reference to this question, that of *Drosophila*,[26] there is evidence that such "sectional deficiencies" do comprise a good deal larger proportion of the point mutations obtained by irradiation of the mature germ cells than of the point mutations arising naturally. However, the apparent point mutations produced by irradiation of immature germ cells of *Drosophila* do not include substantially more that on further analysis prove be be demonstrable "sectional deficiencies" than are found among the natural mutations. Moreover, the characteristics of the effects produced on the individual, both in *Drosophila* and mice,[10,27] also indicate that a large proportion of these radiation-mutations are as truly changes within the genes as are the mutations of natural origin.

In general, then, in the animal material, the radiation-mutations strongly resemble the natural ones. Practically all types of natural point mutations that have been looked for in extensive irradiation experiments have been found to be produced by radiation also. Like natural mutations, of course, the great majority, although not quite all, of those produced by radiation, are detrimental. Moreover, the great majority have far less dominance (i. e., less expression in the heterozygous individual) than the normal genes from which they arose. Once arisen, the radiation mutations, like the natural ones, are permanent, reproducing themselves as such.[28]

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[10] See footnote, p. 1071.

[24] J. B. S. Haldane, Amer. Naturalist 71: 337–49 (1937). (Volume and year given through typographical errors as 11 and 1949 in original. Note added Oct. 5, 1955.)

[25] L. J. Stadler, Cold Spring Harbor Symposia Quant. Biol. 9: 168–77 (1941). L. J. Stadler, Science 120: 811–19 (1954). L. J. Stadler and H. Roman, Genetics 33: 273–303 (1948).

[26] H. J. Muller, J. I. Valencia, and R. M. Valencia, Genetics 35: 126 (1950).

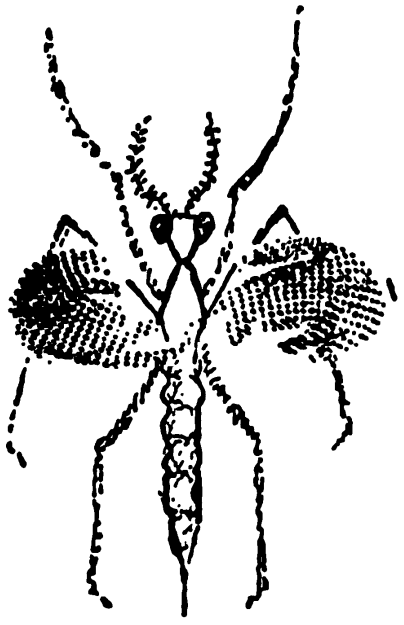
[27] H. J. Muller, "Gene Mutations Caused by Radiation," Symposium on Radiobiology, John Wiley & Sons, New York, Chap. 17, pp. 296–332 (1952).

[28] H. J. Muller, Cold Spring Harbor Symposia Quant. Biol. 9: 151–65 (1941).

Just which mutation is produced by radiation on a given occasion is of course a matter of "accident," as is true of natural mutations. However, the total frequency of the mutations produced by a given dose of radiation varies to some extent with the accompanying conditions, as in the case of natural mutations, although the conditions in question are to some extent different ones in the two cases. The conditions which influence the production of point mutations by radiation include genetic differences, differences in cell type or stage, differences in metabolic reactions, and (a category overlapping the previous one) differences caused by the application of special chemical or physical treatments.

For the most part, the same influences have been found to promote or hinder the action of radiation in causing point mutations as in causing structural changes of chromosomes. For example, chromosomes in condensed stages are more susceptible to the induction of changes of both types. Some findings of interesting differences in this respect have been reported, however. Among these are the observations that sperm cells of *Drosophila* several days prior to their release, and therefore perhaps in the spermatid stage, are much more susceptible than mature spermatozoa to the production of radiation of structural changes, but not of point mutations.[30]

In accordance with the view, first proposed by Rapoport[30] on the basis of chemical work by Fricke,[31] that the mutagenic action of radiation is exerted via the production of actively oxidizing radicals or molecules, it is found that radiation mutagenesis of both major types is positively correlated with the amount of free oxygen present at irradiation. Physical or chemical influences which appear directly or indirectly to increase or decrease the abundance of oxygen available for conversion into mutagenic radicals influence correspondingly the frequency of mutations produced. There is, to be sure, evidence indicating that not all the mutagenic action of radiation takes the same pathway, and that some of it may be quite unconnected with oxidation. But, however that may be, the above and other findings, by demonstrating the conditional nature of radiation mutagenesis, constitute a disproof of the target hypothesis of such mutagenesis, at least in the simplified form in which it had sometimes been applied.[32] Moreover, these findings are of considerable practical value in having led to the working out of treatments, described in other sections of this conference,[33] which give hope of affording significant protection against the mutagenic action of radiation. The fact that certain treatments, even when given after irradiation aid in such protection, is especially noteworthy, both from a theoretical and from a practical standpoint.



[30] K. G. Lüning, Acta Zool. 33 : 193–207 (1952).
[30] J. A. Rapoport, Zhur, Obshchei Biol. 4 : 65–72 (1943).
[31] H. Fricke, J. Chem. Phys. 2 : 556–57 (1934). H. Fricke, Cold Spring Harbor Symposia Quant. Biol. 3 : 55–63 (1935).
[32] H. J. Muller, J. Cellular Comp. Physiol. 35 (Suppl. 1) : 9–70 (1950).
[33] A. Hollaender, Science 121 : 624 (1955). A. Hollaender, W. K. Baker, and E. H. Anderson, Cold Spring Harbor Symposia Quant. Biol. 16 : 315–26 (1952).

In material of varied kinds, but more especially in *Drosophila*, there is good evidence that over a considerable range of dose (in *Drosophila*, from some 50 r. to more than 1,000 r., a more than twentyfold range) the frequency of point mutations (like that of chromosome breaks) is directly proportional to dose.[34] Moreover, they are independent of the timing of the dose, over an enormous range, provided cellular conditions are held constant.[35] Below 25 to 50 r. the mutation frequency is so low that it has hitherto been impossible to obtain sufficient data, and above 1,000 or 2,000 r. the determination of frequency may be interfered with by a selective elimination (through chromosome aberations) of the cells that happened at irradiation to be in a more susceptible state.[36] Since, however, in the work with low doses and low time-rates of delivery of gamma radiation the germ cells of some series were traversed by only one electron track in a period of a half hour or more, on the average, and still showed a frequency of mutations proportional to the total dose, there is reason to infer that no dose or intensity of such radiation is without its proportionate production of point mutations. Moreover, if this is true of gamma radiation it must be at least as true of radiation producing tracks more densely crowded with ionizations.

Despite the equal mutagenic efficiency of different doses and dose rates of ionizing radiation, it is not necessary to infer that a point mutation or a break is ordinarily the consequence, direct or indirect, of a single activation or even of a single ionization. For all the ionizing radiation studied, has some of its ionizations produced in clusters of minute diameter. If two or more ions commonly cooperate mutagenically, however, it might be thought that this would become evident by causing the frequency of mutations to vary as the square or some higher power of the dose. Yet this would not be true if those ions had to be as near together as the ones in a natural cluster, for such close juxtaposition as this would not be brought about with appreciable frequency by raising the dose and the dose rate within toleration limits.

That such cooperation in mutagenesis does occur is indicated by recent observations to the effect that fast neutrons appear to be approximately twice as efficient as X or gamma rays in inducing point mutations in the chromosomes of *Drosophila* spermatozoa,[37] and are probably a good deal more efficient still, relative to X or gamma rays, in inducing chromosome breaks.[38] Presumably alpha rays likewise would be more efficient than X or gamma rays in these respects. One possible interpretation of this higher effectiveness of fast neutrons would be provided, on the Watson-Crick hypothesis of the structure of the genetic material, by the doubleness of the fibers in which the rearrangements are produced, if we suppose that the occurrence of the mutation or break is much facilitated when both fibers are simultaneously affected.

The effectiveness of fast neutrons in inducing point mutations is actually higher than it appears to be, because intensive studies of given cases of these seeming point mutations have shown that in fact a considerable proportion of them involve a double or multiple effect within a very localized chromosome region.[39,40] This greater clustering of effects with neutrons than with X or gamma rays is to be expected, in view of the greater concentration of the ionizations in the tracks of the ionizing particles released by neutrons, provided that the mutational effects arise in close proximity with the activations that induce them. Since this clustering of effects causes many of them to be lost to

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[34] C. P. Oliver, Z. indukt. Abstamm. u. Vererbungslehre 61; 447–88 (1932). W. P. Spencer and C. Stern, Genetics 33: 43–74 (1948). N. W. Timofeeff-Ressovsky, Experimentelle Mutationsforschung in der Vererbungslehre, T. Steinkopf, Leipzig (1937).

[35] K. G. Lüning, B. Lindell, and R. Falk, Acta Radiol. 43: 89–92 (1955). J. T. Patterson, Biol. Bull. 61; 133–38 (1931). S. P. Ray-Chaudhuri, Proc. Roy Soc. Edinburgh B62: 66–72 (1944). N. W. Timofeeff-Ressovsky and K. G. Zimmer, Strahlentherapie 53: 134–38 (1935). D. E. Uphoff and C. Stern, Science 109: 609–10 (1949).

[36] H. J. Muller, I. H. Herskowitz, S. Abrahamson, and I. I. Oster, Genetics 39: 741–49 (1954).

[37] P. T. Ives, R. P. Levine, and H. T. Yost, Jr., Proc. Nat. Acad. Sci. USA 40: 165-71 (1954). G. H. Mickey, Amer. Naturalist 88: 241–55 (1954). H. J. Muller, Rec. Genet. Soc. Amer. 23: 58 and Genetics 39: 985 (1954).

[38] W. K. Baker and E. Von Halle, Science 119: 46–49 (1954). A. D. Conger, Science 119: 36–42 (1954). J. S. Kirby-Smith and C. P. Swanson, Science 119: 42–44 (1954). W. L. Russell, Liane B. Russell and A. W. Kimball, Amer. Naturalist 88: 268–86 (1954).

[39] H. J. Muller and J. I. Valencia, Rec. Genet. Soc. Amer. 20: 115–16; and Genetics 36: 567–68 (1951).

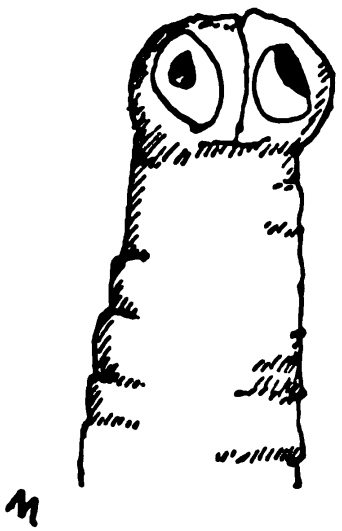
[40] H. J. Muller, "The Manner of Production of Mutations by Radiation," Radiation Biology (edited by A. Hollaender), McGraw-Hill Co., New York, Vol. I, Chap. 8, pp. 475–626 (1954).

view by reason of their proximity to each other (except when special techniques of analysis are used), the mutagenic potentiality of the fast neutrons is correspondingly underrated in most experiments. So far as genetic damage to the population is concerned, however, a double or multiple effect of the given kind adds no more to the mutational load than does a single effect. Hence for present purposes fast neutrons may be regarded as no more than twice as effective as X or gamma rays in producing point mutations.

ESTIMATION OF THE TOTAL POINT MUTATIONAL DAMAGE FROM A GIVEN AMOUNT OF RADIATION

It has been noted that the important quantity in the determination of the total amount of genetic damage is not the amount of harm done to the individuals who have inherited the mutations in question but only the total number of these mutations. For a mutation doing less harm to an individual will, as if in compensation, be passed down to a correspondingly larger number of descendant individuals. It has also been noted that an approach to a direct estimation of the total number of mutations arising has thus far been made only in *Drosophila*, and that this calculation has involved the use of data from radiation experiments. This work can therefore be applied to the estimation of the total damage arising from a given dose.

The principles have already been explained whereby a minimum value for the total number of mutations is obtained by getting the number of lethals in the X chromosome and then multiplying this by 6, to get the number of lethals in all the chromosomes, and again by 4, to get the total number of mutations causing at least 10 percent detriment to life, when homozygous. (A correction is made in this calculation, based on certain tests, in order to estimate the number of point mutations without including the structural changes.) When this calculation is carried out, using the results obtained at any given dose, the resulting number can then be expressed in terms of the total number of point mutations produced by a single r. unit, by using the principle of proportionality of point-mutation frequency to dose. It is then found that this number turns out to be about 1 mutation among 2,000 germ cells per r. (that is 5×10^{-4}/r.) for X or gamma rays applied in the usual way to mature spermatozoa.[41] The more important figure, representing the result of irradiation of the more prevalent stages (gonia) of immature germs cells of adult *Drosophila*, is only a fourth to a half of this, according to the conditions. It is probable that there are even lower values for certain other immature stages of *Drosophila* germ cells, as for instance those in the embryonic polar cap.[42]



In order to obtain a figure for the total number of mutations produced by a given dose in mammalian material we may follow the procedure which we

[41] H. J. Muller, "Radiation Damage to the Genetic Material," Science in Progress, Yale University Press, New Haven, pp. 93–165, 481–93 (1951). H. J. Muller, "The Nature of the Genetic Effects Produced by Radiation," Radiation Biology (edited by A. Hollaender), McGraw-Hill Co., New York, Vol. I, Chap. 7, pp. 351–473 (1954).

[42] Z. I. Berman, Izvest. Akad. Nauk, SSSR, pp. 645–78 (1939). Helen U. Meyer, unpublished data.

adopted in the calculation of natural mutations. This involved a comparison of the mutation frequencies involving particular types of mutations, located in given positions on the chromosomes, in *Drosophila* and in mammals, and then applying the ratio thus found to the figure for total mutation frequency in *Drosophila* so as to convert it into the presumed corresponding value (a minimal one) for mammals.

Fortunately, there is available for this comparison a much more reliable body of data, for both groups of organisms, than that which we had recourse to in the case of natural mutations. The average frequency of point mutations of the kind in question in *Drosophila*, based on a study of 10 types (loci), was found to be about 1.4×10^{-6}/r. for any given type, when the radiation was applied to inactive immature germ cells (oögonia).[43] The different types seldom varied from one another in frequency by a factor of more than 2 in material abundant enough for judging this matter (that in which spermatozoa had been irradiated.

In the mammalian material, comprising irradiated spermatogonia of mice, Russell[20] has reported an average mutation frequency of about 25×10^{-6}/r., based upon 7 specific types of mutations (loci). Here the range of variation between the different types was greater than in the above *Drosophila* material, but their mean agreed well with their mode, and 4 of the 7 types conformed fairly closely with this mean. It is clear on comparison of the two sets of results that the susceptibility of the mammalian material is at least an order of magnitude higher than that of the flies, the observed factorial difference in results being 18. To obtain a minimum estimate of the total frequency of mutation in the mice we must therefore multiply by 18 the figure arrived at for gonial cells of flies. (It is of no consequence that in the flies oögonia were studied and in the mice spermatogonia, since special comparisons[44] have shown these two cell types to be alike in mutagenic susceptibility as they are expected to be.) Since the figure for the gonia of flies had a lower limiting value of 1.25×10^{-4}/r. the minimum value for mice becomes 2.25×10^{-3}/r. This is the frequency for a germ cell, not for an offspring derived from two such germ cells; for the offspring it would be 4.5×10^{-3}/r.

In performing this calculation we are, as in the case of the natural mutations, assuming that the hereditary material of mammals is no more compound than that of flies; i. e., that there is not a greater number of different specific types of mutations in mammals than in flies, despite their seemingly more complicated organisms and their larger amount of deoyribonucleic acid. The total frequency of mutations per r. may be a good deal higher than here calculated not only because of the inadequacy of this assumption but also because weakly detrimental mutations and those mainly affecting fertility rather than individual survival have not been included. Moreover, only the lower limiting value for the somewhat variable mutation frequency of fly oögonia was used. All this emphasizes the fact that our estimate is decidedly on the "conservative" side.

At the same time, it is true that the value is one for mice, not human beings. All that can be said to this is that, so long as we lack data on an organism still closer to man, it is necessary, provisionally, to base our judgments on this result, and that, since mice are so much closer to men than flies are in almost every other important respect, it would be strange if they were not closer in their mutagenic properties as well. Moreover, the factors which might be expected to cause a significant difference in the natural mutation frequencies of mice and men—their great discrepancies in length of life, size, and number of cell generations in the reproductive cycle—would not be expected to exert significant influences on the frequency with which mutations are produced in them by radiation.

The minimum figure of 4.5×10^{-3}/r. point mutations for the offspring of parents both of whom were exposed can be expressed in the form: "at least 1 induced point mutation per offspring, on the average, for each 220 r. of exposure to both parents. From this it is evident that many of the children who were conceived by Hiroshima survivors at any time after their exposure must have contained one or more mutations induced by the radiation. Similarly, children conceived

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[20] See footnote, p. 1075.

[43] H. J. Muller, Seventh Annual Report to Amer. Cancer Soc., Inc., pp. 120–21 (1952).

[44] K. V. Kossikov, Genetics 22: 213–24 (1937). R. I. Serebrovskaya and N. I. Shapiro, Compt. Rend. Acad. Sci. U. R. S. S. 2: 421–28 (1935).

NOTE.—Only when these two papers are taken in conjunction with one another does the equal mutagenic susceptibility of gonial cells of the two sexes become evident.—H. J. M.

by parents both of whom have been exposed to the so-called "permissible dose" of 0.3 r. per week (15 r. per year) for as long as 15 years would on the average contain at least 1 induced mutation. It is probable that the same is also true of the children of many radiologists, dermatologists, and dentists.[45]

The recent study of Macht and Lawrence,[46] gives direct evidence of genetic damage in such cases and is in this respect superior to the studies made in Japan. Moreover, studies of Moeller et al.[47] show that the population in general is already receiving significant amounts of radiation from medical diagnoses. Sonnenblick [48] finds that exposures of this kind are seldom adequately controlled.

When it is considered that practically every mutation must eventually become eliminated from the population, after having—even if imperceptibly—hampered enough descendants so as finally to be a deciding cause, in the last of the line, of his premature death or failure to reproduce, then it becomes evident that practically every mutation represents a postponed disaster. Thus the genetic damage, that to later generations, caused by a given total dose is seen to be far greater than the damage to the exposed individual himself. In view of this, measures and regulations concerned with radiation protection should be based, at least in the case of persons who may later reproduce, primarily on the risk of genetic damage or, more specifically, of point mutations in their germ cells, rather than on the risk of damage to their own bodies. This would cause such measures and regulations to be far more stringent than they are at present.[49]

THE INDUCED MUTATIONS IN RELATION TO THE NATURAL LOAD

On our conservative estimate of 16 million natural mutations arising per generation in a population of 100 million, a frequency of 0.16, it would take only about 37 roentgens of gamma radiation delivered to the population to produce a quantity of new mutations equal to the new natural ones, and thus to double the mutation frequency. Our conservative estimate, however, was based on the assumption of only 1×10^{-5} as the average freqeuncy of a mutation of some specific type, involving a given chromosomal position. According to this assumption the actual data, which indicate about 2×10^{-5} as the frequency of mutations of a specific type, are misleadingly high, because of certain sources of technical error. Since, however, this is a matter by no means proved as yet it remains quite possible that the amount of radiation necessary to double the mutation frequency is 75 roentgens or higher. This is approximately the value that we used in our earlier treatments of the subject,[49,50] in which it was assumed that the observed 2×10^{-5} frequency for mutations of a specific type was approximately correct. These considerations illustrate the considerable margins of error in any present quantitative treatments, and the need for greater exactitude of knowledge.[51]

The present uncertainty regarding the natural mutation frequency carries with it a corresponding uncertainty regarding what proportion of the natural mutations in man are contributed by natural radiation. There is also uncertainty regarding this question based on variation in the amount of natural radiation. If we suppose that in some typical regions as much as 6 roentgens are accumulated, on the average, in the span of one human reproductive generation (25 to 30 years), then, on the more conservative estimate that the natural mutation frequency is equal to what would be induced by 37 roentgens, it turns out that some 16 percent of the natural mutations in man are produced by natural radiation. On the higher estimate for natural mutations, some 8 percent of them would be radiation-induced. In either case, the figure must be far higher than for short-lived organisms, such as mice or flies. On the other hand, in some

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[45] H. J. Muller, Bulletin of the Atomic Scientists 11: 210–12, 280 (1955) and Science 121: 837–40 (1955).
[46] S. H. Macht and P. S. Lawrence, Amer. J. Roent. and Rad. Ther. 73: 442–66 (1955).
[47] D. W. Moeller, J. G. Terrill, Jr., and S. C. Ingraham, II, Public Health Rep., U. S. Publ. Health Serv. 68: 57–65 (1953).
[48] B. P. Sonnenblick, J. Newark Beth Israel Hosp. 6: 81–42 (1955).
[49] H. J. Muller, Acta Radiol. 41: 5–19 (1954).
[50] H. J. Muller, Amer. J. Obstet, and Gynec. 67: 467–83 (1954).
[51] Much of the information concerning mutation frequencies in Drosophila cited in the present report was derived from work by the present writer and his associates that had been supported by grants from the Atomic Energy Commission (contract AT (11-1)-195), the American Cancer Society (given on recommendation of the Committee on Growth of the National Research Council, United States of America), and the Rockefeller Foundation.

human populations living at a high altitude, with its greater cosmic ray intensity, the contribution of radiation to the natural mutation rate must be twice as high as here estimated. Still higher values must obtain for some populations living in regions where radioactive minerals are abundant.

Many persons unfamiliar with genetics have regarded the seeming normality of the children born to survivors of the Hiroshima and Nagasaki bombings as evidence against the conclusion that the amount of radiation there received produced a significant amount of genetic damage. This misunderstanding arises from their lack of realization of the following points.

1. Few mutations are sufficiently dominant to give readily perceptible effects when inherited from only one parent, as they are in the vast majority of cases.

2. Even though these effects are not perceptible they are nearly always sufficient to hamper the individual somewhat, and finally, usually in a very distant descendant, to cause the extinction of that line of descent.

3. In any heterogenously breeding population, such as is found anywhere outside of the geneticists' fields and laboratories, there is already so much natural genetic variation, representing an accumulation of many generations of natural mutations, that the additional mutations caused by the radiation would become lost to view among them even if they were as abundant as those that would arise naturally in the course of a number of generations. Thus, the genetically damaged population will eventually have to pay the costs, but these will be spread out over so many small installments, and so intermingled with the greater weight of other payments, as hardly to be recognizable. All this was of course well known to geneticists before the observations on the children at Hiroshima and Nagasaki were conducted, and led them to express serious doubts that any genetic effects would be demonstrable there, even though they had no doubts that they had actually been produced.[41]

These points may be better appreciated if it is realized that in *Drosophila* also it had not been possible to demonstrate the mutagenic action of radiation by mere inspection of the individuals of the first, second, or third generations after exposure. Exact genetic methods had first to be worked out[11] and these are of course unavailable in man. Even following an exposure of fly spermatozoa to some 5,000 roentgens, which we today know causes each offspring, on the average, to receive at least three induced mutations, hardly one abnormal offspring is usually to be found among 100 examined, yet the damage is there, and it will be exerted if the population is allowed to continue.

At the same time, it is true, unlike what many nongeneticists suppose, that the effects of the genetic damage are more strongly exerted in the first generation of offspring than in any subsequent generation. They very gradually subside, in the course of many generations, as the population is purged by the dying out of the unfit. Even the recessive effects, those present in individuals homozygous for the given mutations, are found most frequently in the first generation, and then less and less frequently if the population breeds naturally rather than being subjected to a geneticist's controlled inbreeding manipulations. Moreover, there is a much higher chance that a given induced mutation will become homozygous by meeting, at fertilization, a gene of the same type derived from the great accumulated store of natural mutations, than one of that type which, like itself, had been induced by the radiation.

Since the worst effects are already exerted in F_1, what the Hiroshima observations do demonstrate clearly is that the genetic damage to posterity caused by exposure to between one hundred and several hundred roentgens is not *conspicuously* detrimental, and is well within limits consistent with the survival and self-perpetuation of the population. This might have been reckoned as probable without the direct evidence. For, according to the conclusion that the average individual is already heterozygous for some 6, 8, or even more mutations which when homozygous would be fairly conspicuous and/or detrimental, it does not seem likely that the addition in heterozygous condition of just one more, induced by some 200 roentgens, would result in any very evident change in the picture. This remains true even when we take into consideration the fact that the already existing mutations have already passed, to varying extents, through the sieve of selection and are therefore not, on the average, as detrimental as the newly induced ones.

The apparent contradiction between the fact that a really serious amount of genetic damage was produced and the fact that none is evident even in the most

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[41] See footnote, p. 1079.
[11] See footnote, p. 1072.

afflicted generation (the first), is reconciled by the manner in which the damage is spread out, thinner and thinner, over a great number of generations. There is a kind of buffering or dilution of the damaging effects, by the normal genes that dominate over them and thus delay their elimination. Thus the effects are spread out in time, in inverse proportion to their dilution in any one generation, but the total damage remains just as great as if concentrated. Moreover, even though the induced mutations may be many times the number that would arise naturally in one one generation, they are nevertheless few in relation to the accumulated natural "load." Hence they can raise by only a rather small percent the number of genetic shortcomings already present in the population.

Despite these buffering influences, it would be impossible for a population to tolerate, generation after generation, an exposure which, given to only one generation, would cause no perceptible deterioration. Gradually, as elimination rose enough to balance the new mutations, an equilibrium level of accumulation would be approached, and at this new level the then existing accumulated load would be as many times greater than the original accumulated load as the then existing mutation rate was greater than the original mutation rate. Thus, if 37 roentgens doubles the mutation rate, a population which had received this dose for many generations would at last have twice as many ills of genetic origin as we have. Yet we already have more than enough for comfort.

Not to be neglected in the picture is the other end of the balance mechanism; the rate at which elimination of mutations goes on. Under modern civilization we interfere so much with this that we are probably raising the load of accumulated mutations as fast as by applying some tens of roentgens to everyone's reproductive organs.[43] Under these circumstances the raising of mutation frequency at the same time, by exposure to radiation, might tend to bring us to a genetic situation that it would be difficult to cope with.

All these questions need to be not only discussed but actually investigated far more realistically than they have been in the past. Otherwise we may at last find ourselves, genetically, facing a parallel to already accomplished deforestation and erosion, on an even grander scale. This problem is not only one that is concerned with the possible aftermaths of atomic war. It must be faced equally by the proponents of peace if we are to have an atomic age, with its risks of prolonged "permissible" exposures arising from industrial uses and radioactive waste products.

For peace will, we hope, go on and on through a great series of generations. Under these circumstances, it will be the more necessary to control and limit the radiation received by the population at large in every generation. For, given enough generations, the equilibrium level of damage will be reached, at which that damage will no longer be buffered, but will accurately correspond with the existing mutation frequency. Then, a relatively small number of roentgens per generation will exert an inordinately larger effect than it seems to now. At our present juncture, before that process has more than begun, far-seeing policies should be established. These must guard us against the dangerous fallacy that what cannot be seen or felt need not be bothered with.

This subject of protection of human beings against the genetic damage produced by radiation must, until suitable policies are established, far overshadow in its importance that of the utilization of radiation in the genetic improvement, for human purposes, of organisms potentially useful to man, or in the elimination or reduction of noxious organisms. However, these constructive uses of radiation in biological engineering will come increasingly to the fore as the more menacing aspects of radiation are brought under control. There is already abundant evidence of the possibility of such beneficial applications on a considerable scale.[41] [42]

At the same time, the dangerous mistake should not be made of considering man as a species who would himself undergo a long-term benefit from the application of radiation to his germ plasm. His own reproductive material is his most invaluable, irretrievable possession. It is already subject to an amount of variation which, in relation to his present reproductive practices, borders on the excessive. Under these circumstances, man's first concern in dealing with radiation must be his own protection.

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[41] See footnote, p. 1079.

[42] A. Gustafsson, Cold Spring Harbor Symposia Quant. Biol. 16: 263–281 (1952). A. Hollaender, Ann. Missouri Botan. Garden 32: 165–178 (1945).

Representative HOLIFIELD. Are there any questions by members?

Representative VAN ZANDT. Dr. Muller, in regard to Russia, can you estimate the number of years it will take Russia to catch up with our progress in this field of radiation?

Dr. MULLER. I believe it will probably take them a long time. One would have to assess political factors there which are essentially unpredictable. Personalities play such a large role there, much larger than here, despite their theory of economic determination. Anyway, my estimate would be 10 or 15 years.

Representative HOLIFIELD. Thank you very much, Dr. Muller. Mr. Hollister wishes to ask you some questions.

Mr. HOLLISTER. Dr. Muller, I understand that you are familiar with the work of the Atomic Bomb Casualty Commission, and I wonder if you would comment on it and on the quality of and the conclusions from the data.

Dr. MULLER. I think it was evident to geneticists from the beginning that it was very doubtful whether any positive or essentially negative evidence could be obtained from a study of that kind, because human populations are so variable and it is so next to impossible to obtain two groups to compare, one irradiated and the other not, which are essentially similar in other respects. In view of that, I think that the present lack of results is not surprising even to most of those who took part in the investigation. I do not think they are to be blamed for it. There was some slight chance—because of the uncertainty of the estimates of the human mutation rate—that if the induced mutation rate was exceptionally high then some evidence of it could be obtained.

I think that it is very unfortunate to cite the lack of results from the study as indicating that there is no effect. I think that those reporting on the matter for the AEC in a December 1956 publication that I recently saw were quite right in saying that the data obtained there are not at all out of line with the expectation based on results with mice, that is, the frequency of induced mutations could well have been just as high among the human beings for the dose received as it would have been for mice, which is a matter we know a lot more about through Dr. Russell's experiments. We can go a little further than that and say that it could not have been a great many times higher than it is in mice, otherwise there would have been demonstrable results.

But we need not feel at all secure or relieved that no effects were found.

I remember that in a meeting of a committee called to decide whether these studies should continue one of the persons on the committee said, after I had remarked that I thought no effects would be found and that that would not prove anything, "That will be a very good thing because it will tend to allay the public fear on the matter."

Chairman DURHAM. Did you participate in writing the report?

Dr. MULLER. No; I did not. I may say this: I do disagree with a lot of the discussion in the last chapter where exception is taken to drawing conclusions from the results obtained in lower organisms. Certain criticisms are leveled against the work on flies and mice, for example, that I think are demonstrably unjustified and have a mistaken position. But that is not the major part of the work. When it

comes to the work on humans, I think that was done as well as could be expected under the very difficult circumstances.

Representative HOLIFIELD. Are there any further questions?

Representative VAN ZANDT. Dr. Muller, in the December 3, 1956, issue of the Federation of American Scientists you are quoted as saying this:

> It is reckless to increase the risk of war by continuing H-bomb tests. It is not the fallout from these tests that is at issue at this time but the war feeling. The first step for peace open to us is a discontinuance of tests by both sides. If breached by either side it can be detected by the other.

Will you comment on that statement?

Dr. MULLER. I was not speaking there primarily as a geneticist, but I think physicists are pretty generally agreed that the setting off of a test in the megaton range can be detected with certainty by the other side. So that a breach would be known and in that sense we already have 100-percent-effective inspection for tests of that kind.

Therefore, it is in that sense safe if both sides agree to discontinue the tests for them to act upon that agreement, since as soon as one side breaks it, the other side will know it.

Senator ANDERSON. As a matter of fact, Doctor, the first times that we detected tests we detected them with instruments that are primitive compared to what we now have; is that not true?

Dr. MULLER. And we detected ordinary atomic bomb tests of the other side.

Senator ANDERSON. So that a megaton test would be easily detectable, and there is no possibility of deception.

Dr. MULLER. Yes.

Representative HOLIFIELD. Thank you very much.

The next witness is Dr. W. L. Russell.

Dr. Russell comes from Oak Ridge, and has done important work at the Laboratory. We are glad to have you here and to have your statement.

STATEMENT OF DR. W. L. RUSSELL, OAK RIDGE NATIONAL LABORATORY [5]

Dr. RUSSELL. Mr. Chairman, members, the testimony to be given here is presented in response to one of the requests by this committee for scientific results on the biological effects of radiation caused by events other than fallout. Assuming the present estimates of radiation from fallout to be approximately correct, it would in fact be virtually impossible at the present time to measure the genetic effects of fallout in mammals. In estimating the genetic hazards of fallout, we are, therefore, forced into using the information obtained from experiments, such as those to be described, in which much higher levels of radiation were used.

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[5] B. A., Oxford University, 1932; Sherman Pratt fellow, Amherst College, 1932-33; fellow, University of Chicago, 1933-34; asistant, department of zoology, University of Chicago, 1934-36; Ph. D., University of Chicago, 1937; research associate, Roscoe B. Jackson Memorial Laboratory, Bar Harbor, Maine, 1937-47; principal geneticist, Oak Ridge National Laboratory, 1947 to present; research and publications on the genetic effects of radiation in mice. In charge of the Mammalian Genetics and Development Section of the Biology Division of Oak Ridge National Laboratory. Member of the United States delegation to the 1955 Geneva Conference on the Peaceful Uses of Atomic Energy. Member of the Committee on Genetics Effects of Atomic Radiation, National Academy of Sciences. (Submitted by witness.)

The results described here were obtained from a series of experiments conducted in the Mammalian Genetics and Development Section of the Biology Division of the Oak Ridge National Laboratory. This program was started in 1947, the year the Atomic Energy Commission was founded. The preliminary work involved the construction of animal rooms and laboratories, the development and building up of special stocks of mice necessary for the experiments, and certain pilot experiments, including studies on the effects of radiation on fertility. The major experiments were started in 1949.

I was very happy that Dr. Sturtevant said what he did this morning about the lack of suppression of information and the desire to do good work in the national laboratories. I heartily concur, but such a statement comes more forcefully from a person outside the AEC, and one of Dr. Sturtevant's standing. I will say no more on this, other than that neither I nor any of my colleagues would attempt to do scientific work under conditions that were not free.

I think I might also add, and it would be ungrateful of me if I did not, that in addition to working under free conditions and not having our information suppressed, we have received personal encouragement, especially from the Division of Biology and Medicine of the AEC, including its Directors, from Dr. Warren, the first one, to the present one, Dr. Dunham.

Before the results of these experiments with mice were obtained, estimates of genetic hazards of radiation in man were based primarily on data from experiments with the fruitfly, *Drosophila*. Some information on the genetic effects of radiation in mammals was available, but most of this dealt with major chromosomal aberrations which are probably not an important hazard in human exposures. There was virtually no information on radiation-induced gene mutation rates in any mammal. The information now available from our experiments has therefore played a basic role in the new estimates of genetic radiation hazards made and published last year by the National Academy of Sciences committee in the United States and by the Medical Research Council committee in Great Britain. The United Nations Scientific Committee has also used this information.

In response to the request by this committee, I should like to present here a simplified up-to-date summary of our results, emphasizing the aspects which are useful in estimating human hazards. More detailed technical accounts have been submitted for the record to supplement this oral presentation. Again it must be kept in mind that these data were not obtained from fallout radiation. However, they can be used to estimate the genetic hazard from fallout, provided the radiation dose to the gonads is known. I shall describe two types of experiment. One of these measures the rates at which genes mutate by studying the effect of radiation on a particular selected sample of genes. The other measures damage in a population as a result of the total amount of mutation induced.

Most of our work up until recent times has been concerned with the former method, and so I shall describe the results obtained by it in more detail. The measurement of gene mutation rates is important, both for absolute and for comparative purposes. The particular method that we have used was chosen in the hope that it would be a fine-edged tool for making comparisons. One of these that was ob-

viously badly needed was a comparison of the mutation rates in any mammal with those in Drosophila. The mutation rate method chosen by us seemed likely to be the one which would give the most meaningful information on such a species comparison. Our present information on this point indicates that mouse genes are on the average approximately 15 times as sensitive to radiation as Drosophila genes. The earlier estimates of genetic hazards in man based on results in Drosophila have, therefore, been revised.

Another question on which information was badly needed was whether or not there is any recovery from genetic damage with time after irradiation. Some geneticists believed that there would be no such recovery. Others thought that some recovery might occur. Critical evidence on this point had not been obtained. Most of the information along these lines came from experiments with Drosophila sperm, whereas the evidence needed was for immature sex cells. Let me elaborate for a moment on the importance of the cell stage, i. e., immature versus mature germ cells, before we continue with the question of recovery.

In the testis, the immature germ cells—called spermatogonia—persist throughout life. Cells are constantly budded off from them and, after further multiplication, develop into the mature sperm cells. The time required for the development of a mature sperm from an immature spermatogonium is only a few weeks. When we are exposed to continuous or intermittent radiation, the dose received by a cell up to and including the spermatogonium stage is the total dose received over the whole period from conception of the individual up to the time when the spermatogonium starts its final development. This is obviously going to be much greater than the dose received during the few weeks of its final development into a sperm cell. Even with acute radiation received as a single dose, the chance of a fertile mating occurring within a few weeks after exposure is small compared with fertile matings that will occur at later intervals. Thus again it is usually the dose received by the immature cells which will count. Therefore, the problem of genetic hazard of radiation in man relates primarily to the immature germ cells. It is on these cells that our studies of induced mutations in mice have been conducted. This has many important implications, one of which is on the question of recovery, to which we can now return.

How might the question of cell stage affect recovery following irradiation? It is possible to imagine that an immature germ cell in which a mutation has been induced might multiply at a slower rate than a normal cell. This is one example of a possible mechanism by which some recovery from genetic damage might occur. It is obvious that experimental data from mature germ cells in Drosophila could not answer such a problem. Considering the possibility that I have just mentioned, along with others, it was clear to us that the question of recovery with time had to be investigated for immature germ cells. It also seemed important that the problem be examined in an organism with a generation time much longer than that of the fruitfly, which is only 10 days, and in mammalian gonads, which are quite different in their makeup and function from those of insects. Having now obtained this information, we find that in spite of the possible mechanisms by which recovery might have occurred, there

is in fact no evidence of any significant recovery with time after irradiation. The offspring of a mating made a long time after irradiation is just as likely to contain a gene mutation as is the offspring of a mating made shortly after irradiation. I might interject here that I am talking only about irradiated spermatogonia. If we included the sperm, there would be some recovery within a short time after radiation. In human hazards, however, we are concerned, as I have already explained, primarily with the immature germ cells. Thus it would appear that it is the cumulative dose which is important, and this principle is now established for the immature germ cells of a mammal. I have dwelt on this point at some length because I have found it to be one that is often raised in discussion. We are so used to the body being able to recover from various types of damage that it is quite reasonable to require rigorous proof that this does not occur with genetic damage.

Another problem that was investigated by our method for measuring gene mutation rates is the relation between mutation rate and dose. On the basis of experiments with other organisms, a linear relation was expected. In other words, it was expected that mutation rate would be directly proportional to dose, that, for example, doubling the dose would double the mutation rate.

Dr. Crow this morning assumed this principle and stated that he was basing his assumption on Drosophila. Therefore, I think it appropriate to state what information we have in this respect in the mouse.

Our first data came from males exposed to 600 roentgens. As the data started to come in from males exposed to 1,000 roentgens it became clear that the mutation rate was significantly lower than expected on the basis of the 600 roentgen results. It seemed likely that this result, which was unexpected on the basis of Drosophila results obtained up to that time, might be due to the fact, already emphasized, that we were dealing with immature germ cells (spermatogonia) in the mice, whereas Drosophila results had been obtained from mature germ cells. If, for example, there were differences in sensitivity to mutation induction correlated with sensitivity to killing of the cells, then at the higher doses the mutation rate observed might represent only the mutation rate for the surviving and more resistant cells. Careful studies in our laboratory by Dr. Oakberg on the amount of killing of spermatogonia with various doses of radiation support this possibility.

Whatever the explanation might be, it was clear that it was important to obtain data on mutation rates at doses lower than 600 roentgens. Since the relation between mutation rate and dose has been found not to be directly proportional above 600 roentgens, it was possible that it would also not be proportional below 600 roentgens. We have therefore begun experiements at lower doses. The data so far obtained from a 300-roentgen experiment show no significant departures from proportionality with the 600-roentgen results. More data are, however, needed.

Representative HOLIFIELD. At that point, Dr. Russell, could you tell this committee how near you think the experiments on mice would correspond to the radiation of human beings? You have made the statement that the mouse is 15 times more sensitive than the fruitfly. Can you give us a comparison between the mice and the human being?

Dr. RUSSELL. I have prepared a short statement involving the whole problem of extrapolating.

Representative HOLIFIELD. Is that contained in the statement?

Dr. RUSSELL. It is not in the typed copy you have, but I shall be glad to read it.

Representative HOLIFIELD. Or could you give me a summary right now?

Dr. RUSSELL. I believe a summary is the best we can do at the present time. I might explain a little more about this dose relation, if I may, on the board.

If we put the 600-roentgen mutation rate at this point here, the 1,000-roentgen result instead of being up here, as expected from the linear relation found in Drosophila, turned out to be down here [pointing on blackboard].

Representative COLE. Doctor, would you go back to the microphone and repeat what you said, because I do not understand it.

Dr. RUSSELL. The first result we got was with the 600-roentgen experiment and is shown here. The points immediately above and below it that I have connected with the line represent the 95-percent confidence interval for this result, that it, with 95 percent probability the true value should lie within that range. The 1,000-roentgen result was expected, from the linearity found in Drosophila experiments, to be correspondingly higher than the 600-roentgen, but actually came out lower. In other words, the relation is not linear. This raises the question of why there is linearity in Drosophila and not in the mouse.

As I have said, we felt that perhaps this lack of linearity in the mouse was due to the killing of the spermatogonial cells themselves, and that at the higher dose the result represents the mutation rate of more resistant cells which were not killed. At the lower dose you get a mutation rate for more sensitive cells, some of which have not been killed.

The question arises what is the curve going to do below 600 roentgens? You can no longer predict that it will be linear between 600 roentgens and zero. There is now some likelihood that it will be higher than expected on a linear basis. Up to this time in the hearings we have had a good deal of discussion of whether the mutation rate is linear and if whether some somatic effects are linear or whether they go up this way, that is, concave upward. These mouse mutation results represent something going this way, that is, convex upward. In other words, the effect at lower doses might be higher than expected on a linear basis. It was obviously important to obtain data at the lower doses. The 300-roentgen result looks like this. At the present time it falls close to the straight line drawn between the results for the 600 roentgens and the zero doses, but as I say, I think we need more data on this point.

Representative HOLIFIELD. This would indicate, then, if this theory becomes established, that the lower dose rates would cause more mutations in the germ cells than the higher rate.

Dr. RUSSELL. This is a possibility raised by this departure from linearity at high doses.

Representative HOLIFIELD. That is because the higher dose kills the cell.

Dr. RUSSELL. That is right. The amount of killing of cells is quite high so it is still possible to get some departure from linearity at lower doses. So we feel that this point needs further experimentation.

Representative HOLIFIELD. But you are not yet ready to set the rate?

Dr. RUSSELL. Three hundred roentgens was the first attempt to provide some information between 600 roentgens and zero. As we go down in the dose, it is much harder to obtain the data because fewer mutations are obtained.

Chairman DURHAM. Are you using only flies?

Dr. RUSSELL. This is all on mice.

I might also interject at this point another comment. A good deal has been said about there being no Drosophila data on mutation rates below 25 roentgens and about there being no mouse data below 300 roentgens. Yet, again referring to Dr. Oakberg's work in our laboratory, he can see and actually measure the killing of spermatogonial cells with doses as low as 2 or 3 roentgens, and even with 1 rep of neutrons. A dose of 22 roentgens kills half of the sensitive spermatogonia. So it seems to me that if we can actually see cells that have been killed by doses as low as 2 or 3 roentgens, and can put this on a quantitative basis, if cells are actually killed at these dose levels, there is no question in my mind that there will be genetic effects from doses as low as this.

Representative HOLIFIELD. This has a tremendous impact on the theory of threshold, does it not?

Dr. RUSSELL. It certainly helps to answer the question of threshold for genetic effects. Of course, the question of threshold for some somatic effects is not answered by this, because killed cells might be replaced by normal cells. So it does not conclusively prove anything about the question of a threshold for somatic effects. I think it supports the already quite well established point of there being no threshold for genetic effects, because here is direct experimental evidence on mammals that cells can be killed measurably by doses as low as 2 or 3 roentgens.

Representative HOLIFIELD. You have been able to observe those?

Dr. RUSSELL. The killed ones can be observed, yes.

Another comparative study that can be made with the gene mutation rate method used by us is on the variation in mutation rates of different genes. Extensive information on seven different genes in the mouse shows a wide range in their mutation rates. The difference between the lowest and the highest rates is more than thirtyfold. This finding is of interest in many respects. For example, it raises the possibility that the so-called rate-doubling dose, the dose required to double the spontaneous rate, might be quite different for different genes.

The results from our gene mutation rate method that I have so far described have been useful primarily for comparative purposes. Thus we have been able to compare mutation rates in mice and fruitflies; we have been able to compare mutation rates at short and long intervals after irradiation; we have compared mutation rates at different doses; and so on. Additional work now going on with this method will, in addition to measuring mutation rates at lower doses, also give information on other comparisons—for example, a comparison between the mutation rates in females and males, a comparison of the

results from long-continued low-level irradiation with those from single dose acute radiation, and so on.

Although designed primarily to provide information for the comparisons that I have already described, the gene mutation study has given some information on the nature and amount of total damage to be expected. First with regard to the nature of the damage, considerably more than one-half of all the radiation-induced mutations obtained from the seven genes studied in detail have proved to be recessive lethals—that is, when an offspring inherits the mutation from both parents, it will die. It seems unlikely that this result is biased in the unfavorable direction, because before the experiments were started it was not known whether or not any of the seven genes chosen for the study could mutate to lethals.

Representative HOLIFIELD. Will you explain that term "recessive lethal"?

Dr. RUSSELL. That is when an offspring inherits the mutation from both parents, it will die. When the organism inherits it from one parent, it won't die. It seems unlikely that this result is biased in the unfavorable direction, because before the experiments were started it was not known whether or not any of the seven genes chosen for the study could mutate to lethals. In other words, the sample of genes chosen was not chosen for production of this type of mutation at all. It was not known whether they could even mutate to lethals.

Some information has been obtained on the time at which these lethals kill the individuals who inherit them from both parents. All the lethals that have occurred at one gene locus kill the offspring at about weaning age. The lethals obtained from another gene locus apparently vary considerably in their time of killing. More information is needed on this point, but it is already clear that a large proportion of the lethals already studied kill at times that would be considered tragedies in human experience. Most of the lethals found do not belong to the category of lethals in which death occurs so early in development of the embryo as to pass unnoticed.

It was anticipated that some, perhaps most, of these lethals would have some deleterious effect when inherited from only one parent. Such deleterious effects have already been observed for some of the lethals. It was expected that these deleterious effects might be observable only in a statistical sense in populations. However, in some cases the deleterious effect of a lethal when inherited from only one parent is large enough to be detectable in the individual. It should be mentioned that a lethal which has a deleterious effect when inherited from only one parent will express its damage in the population far more frequently in this way than in the more drastic effect that occurs when it is inherited from both parents.

As has been mentioned, the results of the gene mutation study can be used to estimate total mutation rate. One way of doing this is to take the information from Drosophila on the ratio of total mutation rate to mutation rate at specific genes and use this to make the calculation of total rate on the basis of mutation rates at specific genes in the mouse. This method of estimation was used in the report of the Genetics Committee of the National Academy of Sciences. Dr. Muller used it before, and I think Dr. Crow incorporated this idea in his calculation this morning.

More direct methods of measuring the overall genetic damage are desirable and one of these is the second of the two methods I should like to describe in this testimony. This method was suggested by the evidence that had begun to accumulate that there are slight dominant deleterious effects of mutations formerly regarded as recessive. Two lines of such evidence came from our work on mice. One has already been mentioned, namely, that at least some of the recessive lethals obtained have some dominant effects. The second line of evidence came from the large populations of animals examined for gene mutations. In that study, the offspring were kept to 3 weeks of age, and it was found that survival to this age was slightly lower in the offspring of irradiated males than in the controls. As a result of these findings, and of evidence from other organisms, it began to seem probable that other effects might be detectable in the first generation offspring of irradiated mice. Shortening of life was chosen as an effect that might reveal, as a statistic of a population, the presence of a variety of minor weaknesses which individually could not be easily detected. Pilot studies on longevity in the descendants of irradiated mice were accordingly started. The results from the first of these were presented before the National Academy of Sciences a few weeks ago, and have just been published.

Dr. Crow did not discuss these this morning. I asked him whether be did not believe them. He said "No," he had left them for me to discuss, and I think he felt it was more appropriate as they are quite recent data.

Representative COLE. When you say that he responded to the question of whether he believed them or not, by saying "No," did he mean he did not believe them, or that was not the reason?

Dr. RUSSELL. That was not the reason.

Senator ANDERSON. Are you going to discuss them?

Dr. RUSSELL. I have submitted for the record the detailed account of this which has been published. I shall be glad to read from this or cite any parts.

Senator ANDERSON. You were here the other day when I read briefly from the news story.

Dr. RUSSELL. Yes.

Senator ANDERSON. Is that what it is based on?

Dr. RUSSELL. Yes.

Senator ANDERSON (reading):

Neutron radiation from atomic bomb can shorten the life of a man's children.

This is quoting from you.

Offspring from a man exposed to such radiation will have their lives shortened on the average of 20 days for each unit of radiation their father has received.

Dr. RUSSELL. Yes.

Senator ANDERSON. We have been talking about receiving up to 400 roentgens. We multiplied that out and came to 8,000 days, and that was 22 years.

Dr. RUSSELL. There are two major qualifications which I believe were included in some of the news reports. I remember that they were in the full Science Service report. These qualifications are first, that we were dealing with neutrons which are probably more effective than X-ray or gamma rays. The other is that we were not dealing with the spermatogonia. In this experiment we deliberately tried to

maximize everything to get the effect on scale so that if there were any such effect, we would detect it. In this we used the maturing germ cells which for mutation rates are at least, shall we say, 2 to 4 times as sensitive as the spermatogonia for straight gene mutation rates. It is possible they are even more sensitive for this effect, but this we don't know yet. A more conservative conclusion from the results is the one I put in the discussion. I think at the present time we would be justified in estimating the effect on the first generation of offspring to be between one-tenth and equal to the effect on the shortening of life of the exposed individuals.

I think it is perhaps fairer to talk about this than the 20 days which, in several respects, is maximized.

Senator ANDERSON. I know. Apparently this article in the Science Service was quoting you. It said, "Dr. Russell found, however, that 'There was a significant effect of radiation on the length of life of the offspring.' In the male mice"—I am sorry, the quotation marks were dropped there. It says the life of the offspring was shortened 0.61 days, I assume, for each unit of radiation.

Dr. RUSSELL. Yes.

Senator ANDERSON. Does that refer to mice?

Dr. RUSSELL. Yes; that refers to mice. A unit of radiation in this case was the rep of neutrons rather than the r. for X-rays or gamma rays. There should be some reduction factor. This is not known. But perhaps it is a factor of about 2.

Senator ANDERSON. Quoting further from this article—

Using this information for human beings, Dr. Russell has figured out that a man's life will be shortened from 5 to 35 days for each unit of radiation received by the father.

Is that a correct statement?

Dr. RUSSELL. That is correct. This again refers to the conditions of this experiment, which are not the conditions of human exposure.

Representative PRICE. What was the unit of radiation?

Dr. RUSSELL. The unit of radiation was a rep.

Representative COLE. A rep is what fraction of a roentgen?

Dr. RUSSELL. A rep is equivalent to the roentgen in its physical effect on tissue, but is more effective in its biological effect for most types of biological effect.

Representative HOLIFIELD. Are you drawing the line between the neutrons which would be received close to a bomb explosion, and the gamma and other types of rays that would be received far away?

Dr. RUSSELL. That is partly correct. There would be a mixture of neutrons and gamma rays. Our experiment was set up only to measure the neutron effects. The animals were shielded behind lead to filter out the gamma radiation. The experiment, as I emphasized in the paper, was not conducted primarily for this particular study. We got the data which showed this rather striking effect, so reported it. We are now conducting experiments designed deliberately to measure the shortening of life. But we felt we should not wait for the completion of these before reporting the effect already observed. However, the qualifications are important, and I must insist that they be understood. Otherwise if you just take the 20-day figure and do not put the qualifications on it, it is probably a much larger effect than would be obtained under normal conditions of human exposure.

Senator ANDERSON. It says—

This shortening of life in the immediate offspring, Dr. Russell warns, in the Proceedings of the National Academy of Sciences, will turn out to be of a magnitude that will warrant serious consideration as a genetic hazard in man.

Is that a correct quotation?

Dr. RUSSELL. That is a correct quotation. I have the same statement in this testimony I was about to read. I do believe that, in spite of the qualifications, this is still a serious effect. I think the 20-day figure is too high. For conditions of human exposure, at least by our present estimates, it should be somewhat lower, but I still think it would be serious.

Senator ANDERSON. From 5 to 35 is a safe figure.

Dr. RUSSELL. No; that would still be under these conditions. If you want a definite figure, I would say at the present time something more like 1 to 10. These are guesses. It is much safer to publish the data you have and put the qualifications on them, than to make guesses about the quantitative values of the qualifications.

Senator ANDERSON. It was pointed out that it could take up to 10 and with 400 r, that would produce 4,000 days which would be 11 years, which would be quite a shortening of human life.

Dr. RUSSELL. Yes.

Senator ANDERSON. Which would be serious.

Dr. RUSSELL. Yes, 400 r. would be serious. To conclude the statement on this, some of which we have already discussed: Our data on this effect are not yet as extensive as we should like. However, though the first sample studied was small, it was sufficient to yield a statistically significant effect which appears to be large and, therefore, of general importance. It should be kept in mind that some of the conditions of this experiment were set up deliberately to increase the chance of obtaining a detectable effect. However, since the effect observed appears to be so large, it seems likely that, even when allowance is made for the conditions of human radiation exposure, shortening of life in the immediate descendants will turn out to be of a magnitude that will warrant serious consideration as a genetic hazard in man.

In conclusion, I should like to emphasize one point in addition to those already expressed in this presentation, a point that Dr. Crow mentioned this morning. The layman tends to think of mutations in dramatic terms as gross monstrosities occurring in the first generation following radiation. Our studies on mice confirm the results obtained from other organisms showing that these are exceedingly rare types of genetic damage. Mutations which cause slight deleterious effects are far commoner.

Before summarizing these points, I should like to say a word or two about extrapolation from mouse to man, the question which the chairman raised earlier. There are, of course, risks in this. I think some of the earlier testimony, from medical workers and others, overemphasized the difficulty. Yet, applying animal results to man is exactly what is done all the time in medicine in the testing of drugs and so on. Others who objected to the extrapolation of mouse mutation data to man had no qualms about extrapolating the rate-doubling dose. I personally would feel safer about extrapolating the induced mutation

rate in mammals from mouse to man than about extrapolating the rate-doubling dose itself.

I will certainly agree that there are some risks in extrapolating on the quantitative points, that is, the actual mutation rates. I think the risks are very much less in extrapolating with regard to the principles or relations between various points. Such principles as the lack of recovery with time, the shape of the dose curve, the relative frequencies of different types of mutations and so on, it seems to me, can be extrapolated with fair confidence.

Some of these are principles that cannot be extrapolated from fruit-flies, because of the biological difference between flies and mammals. Other principles, of course, which were very well established in flies can be extrapolated to mammals. I have been stressing in this report those which I think needed work directly on mammals.

I have listed nine points of this testimony in summary.

1. Present data indicate that mouse genes are approximately 15 times as sensitive to the induction of mutation by radiation as fruit-fly (*Drosophila*) genes.

2. There is no recovery from genetic damage with time after irradiation. This principle has now been established by direct experimental evidence on the material in which investigation was badly needed, namely, the immature germ cells of a mammal.

3. Mutation rates following a dose of 1,000 r. show a significant departure from proportionality with the rates obtained at lower doses. Mutation rates at 300 r. and 600 r. do not as yet show any significant difference from proportionality. However, more data are needed, especially in view of the observed departure from proportionality at the higher dose.

4. Different genes show widely different radiation induced mutation rates.

5. More than one half of the radiation-induced mutations obtained have proved to be recessive lethals—that is, when an individual inherits the mutation from both parents it will die.

6. Most of these lethals do not belong to the category in which death occurs so early in development of the embryo as to pass unnoticed. On the contrary, they kill at times that would be considered tragedies in human experience.

7. The deleterious effect of some lethals when inherited from only one parent is large enough to be detectable in the individual.

8. Rough estimates of the total mutation rate expected from a given dose of radiation have been made from the sample of mouse genes studied.

9. More direct methods of measuring overall genetic damage are being used. The first results from one of these show a significant shortening of life in the first generation offspring of irradiated male mice.

In presenting this statement today, I hope I have given the committee a useful picture of at least a part of the basis on which conclusions on the genetic hazards in man have been reached. Thank you.

Representative HOLIFIELD. Thank you very much, Dr. Russell. I think you have given a very important statement here. It certainly seems to prove that in the case of genetics, at least, any radiation is harmful to the genes.

Are there any questions?

Representative COLE. Mr. Chairman, I do not recall that the doctor responded to your inquiry with respect to the relative sensitivity of human cells in comparison with mice.

Dr. RUSSELL. I think there is some question about extrapolating the exact mutation rate from mice to man. I think there is less risk in extrapolating the principles that we have established in our experiments. However, I think the best we can do at the present time is to use the organism closest to man on which we have data, and that is the mouse. I personally would feel less worried about extrapolating the induced mutation rate from mouse to man than I would about some other extrapolations that have been made.

Representative COLE. Then you are unable to give us any estimate of the ratio between fruitfly, mice, and human being?

Dr. RUSSELL. We have the ratio between mouse and fruitfly. This is 15 to 1. With regard to man and mouse, we have no information other than such as has been discussed. It appears from the Hiroshima and Nagasaki data that man is probably not greatly more sensitive than the mouse. Otherwise more damage would have been observed. However, the results on the Japanese study are fully consistent with a rate as high as has been found in the mouse. I would, like Dr. Muller, quarrel with some of the final conclusions of the report on the study in Japan. I would quarrel with one statement there about this particular point. Even so, in the report on this study, the authors do not raise any question about man being less sensitive than the mouse to radiation-induced mutation. I think the data are not extensive enough to rule out the possibility that he could be more sensitive than the mouse.

Chairman DURHAM. Your second summary point you base on how many years of research, that is, there is no recovery from genetic damage?

Dr. Russell. The first indication came after, I would say, about 2 years' work after the basic experiments were started. This has been further confirmed by more extensive data as time has gone on.

Representative HOLIFIELD. This refers to your experiments on mice.

Dr. RUSSELL. Yes.

Representative HOLIFIELD. Thank you very much, Dr. Russell. We will place your article, Shortening of Life in the Offspring of Male Mice Exposed to Neutron Radiation From an Atomic Bomb, in the record at this point.

(The material referred to follows:)

[Reprinted from the Proceedings of the National Academy of Sciences, vol. 43, No. 4, pp. 324–329, April 1957]

SHORTENING OF LIFE IN THE OFFSPRING OF MALE MICE EXPOSED TO NEUTRON RADIATION FROM AN ATOMIC BOMB[1]

By W. L. Russell, Biology Division, Oak Ridge National Laboratory, Oak Ridge, Tenn.

Communicated by Sewall Wright, January 31, 1957

Introduction.—Only in recent years has evidence begun to accumulate that there are slight dominant deleterious effects of mutations formerly regarded as

[1] Work performed under Contract No. W-7405-Eng-26 for the United States Atomic Energy Commission.

recessive.[2-5] The results to be reported here, and our earlier work on mice,[4, 6, 7] indicate that such effects may add up to an important part, perhaps the most important part, of the genetic hazards of radiation in man. The evidence from the earlier work on mice is that appreciable deleterious effects of radiation become manifest in the first-generation offspring. This evidence is of two kinds. First, work on radiation-induced mutations at specific loci in spermatogonia has shown that among the recessive lethals, which comprise more than one-half of all the mutations recovered, many have dominant deleterious effects which, even for individual mutations, are sometimes large enough to be detected easily. Second, overall population damage was found in the large numbers of animals that are raised as far as 3 weeks of age in the specific loci studies. In all such experiments carried out, the survival to 3 weeks of age is significantly lower in the offspring of irradiated males than it is in the controls. (It should perhaps be pointed out that neither of the above effects, nor the effect reported in this paper, is the result of what the geneticist usually refers to as "dominant lethals," which are major chomosomal aberrations that cause early death of embryos and which, as has been pointed out elsewhere,[8] are probably not an important hazard.)

Our earlier work that showed a significant effect on survival to 3 weeks of age in the offspring of irradiated males led us to expect that there would be measurable deleterious effects later in life. The data reported here show that such is indeed the case. These data furnish a third kind of evidence of first-generation damage and perhaps the most striking one. They were obtained as a byproduct of another investigation, and they are not as extensive as we should like. However, they are the only data we have on this subject that were collected under the expensive and difficult conditions of a field test of a nuclear detonation. Furthermore, although the sample was small, it was sufficient to yield a statistically significant effect which appears to be large and, therefore, of general importance.

Materials and methods.—The material used in the present longevity study was the byproduct of an investigation of the relative effectiveness of neutrons from a nuclear detonation and from a cyclotron in inducing dominant lethals in the mouse.[5] In order to reduce the gamma component of the radiation to a proportion that would not appreciably interfere with the estimation of neutron effects, the animals were shielded with lead. The exposure chambers available were lead hemispheres of 7-inch wall thickness and 14-inch inside diameter. Young adult hybrid males, obtained by crossing inbred 101 strain females with inbred C3H strain males, were exposed inside the hemispheres placed at various distances from the detonation. Control males were placed in hemispheres 2 days before the detonation and for a length of time approximately the same as that required for the exposed animals. Further experimental details are described in the report of the earlier work.[5] One day and a half after the detonation, each male was placed with four adult untreated females of the same hybrid strain. At 18½ days after irradiation each surviving male was placed with a new group of 4 females. Most of the females that became pregnant were killed at a late stage of gestation for the dominant-lethal study. However, since the number of pregnancies turned out to be more than adequate for the dominant-lethal experiment, several of the females were allowed to come to term. It was the offspring of some of these females that were saved for the longevity study described here. All these animals came from matings made from 19 to 23 days after irradiation. A few animals died before weaning, and these were not included in the data reported here. At weaning age the sexes were separated and the animals grouped, so far as possible, six to a cage. They were kept in the same grouping throughout their life span. They were checked at least twice weekly for deaths. Only one animal died at less than 1 year of age, indicating that the conditions under which the animals were kept were good.

The total (neutron plus gamma radiation) dose inside each lead hemisphere was measured, as described in the earlier publication,[5] by means of tissue-

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[2] C. Stern and E. Novitski, Science, 108, 538-539, 1948.
[3] H. J. Muller, J. Cellular Comp. Physiol., 35, suppl. 1, 205-210, 1950.
[4] W. L. Russell, Cold Spring Harbor Symposia Quant. Biol., 16, 327-336, 1951.
[5] C. Stern, G. Carson, M. Kinst, E. Novitski, and D. Uphoff, Genetics, 37, 413-449, 1952.
[6] W. L. Russell, in Radiation Biology, Vol. I, ed. A. Hollaender (New York: McGraw-Hill Book Co., 1954), chap. xii.
[7] W. L. Russell, Proceedings of the International Conference on the Peaceful Uses of Atomic Energy, 11 (New York: United Nations, 1956), 382-383, 401-402.
[8] W. L. Russell, L. B. Russell, and A. W. Kimball, Am. Naturalist, 88, 269-286, 1954.

equivalent ion chambers designed for this purpose at short notice.[9] Subsequently, extensive testing and recalibration of these chambers[10] has led to a revision of the original dose estimates. The doses reported in the present publication are the revised estimates. As was reported earlier,[9] attempts to measure the gamma component of the radiation by means of film dosimeters left a large uncertainty as to the size of this factor. Later tests have been made in which both ionization chambers and chemical dosimeters were used to measure the gamma component inside the lead hemispheres when these were exposed to fission neutrons. According to the latest information,[11] these tests indicate that the gamma-radiation exposure in our experiment was almost certainly less than 10 percent of the total dose.

Results.—The median and mean lengths of life, together with the number of animals, for each dose group are given in table 1. An analysis of variance showed that neither grouping in cages nor sex had a significant effect on length of life. It seems likely that larger samples would show some effect of both of these factors, but as there was no significant effect of them in the present experiment, the data were pooled.

TABLE 1.—*Length of life in the offspring of male mice exposed to neutron radiation 19–23 days before mating (deaths before weaning age excluded)*

Total dose to parent (rep) [1]	Number of offspring	Median length of life of offspring (days)	Mean length of life of offspring (days)
0	103	823	792
81	50	741	754
71	5	717	699
118	22	739	723
136	8	666	688
186	2	756	756

[1] Includes some gamma radiation, estimated to be less than 10 percent of the total dose.

To test whether there was a significant effect of radiation on the length of life of the offspring, the means were fitted to a straight line by the method of weighted least squares. This gives an intercept of 786 days and a slope of -0.609 ± 0.238. Since the residual variance is less than the within-subclass mean square, there is no evidence of nonlinearity over the dose range tested. Even if the true shape of the curve is nonlinear, it will be conservative, in making the test of significance, to assume linearity. The larger mean square was used to compute the variance of the slope, and a two-sided t-test shows that the slope differs significantly from zero at the 1-percent level. If one is willing to accept a one-sided t-test as more appropriate, the significance level is 0.5 percent. Thus there is strong evidence of shortening of life in the offspring of the exposed males.

Discussion.—It is noteworthy that a significant shortening of life was detected in spite of the small sample and the considerable genetic variability that must have been present in a population that was the F_1 of a cross between inbred strains. Furthermore, the weighted mean dose received by the exposed fathers was only moderate, being less than one-sixth of the 80-day median lethal dose as measured from other animals of the same strain exposed under the same conditions at distances closer to the same detonation. While it is true that certain features of the experiment, which will be discussed later, tended to maximize the shortening of life, nevertheless the result observed appears to be so large that it seems quite possible that shortening of life is an effect that might be detectable in studies of the offspring of exposed parents in human populations.

[9] See footnotes, p. 1097.
[9] C. W. Sheppard and E. B. Darden, appendix to J. S. Kirby-Smith and C. P. Swanson, Science, 119, 42–45, 1954.
[10] C. W. Sheppard, M. Slater, E. B. Darden, Jr., A. W. Kimball, G. J. Atta, C. W. Edington, and W. K. Baker, Radiation Research (in press).
[11] G. S. Hurst, personal communication.

TABLE 2.—*Shortening of life in the offspring of fathers exposed to neutron radiation 19 to 23 days before mating—Observed result in the mouse and extrapolation to man (deaths before weaning age excluded)*

	Mouse	Man
Point estimate	0.61 day/r. e. p. to father	20 days/r. e. p. to father.
Lower 95-percent confidence limit	0.14 day/r. e. p. to father	5 days/r. e. p. to father.
Upper 95-percent confidence limit	1.07 days/r. e. p. to father	35 days/r. e. p. to father.

In view of the lack of information on this subject, and specifically the fact that no data of this nature were ready for consideration prior to the writing of the 1956 report of the National Academy of Sciences Committee on Genetic Effects,[12] it is desirable to consider what the present data might indicate when they are extrapolated to man. Taking the estimate obtained from the curve fitted to the mouse data, and assuming that the shortening of life in man would be proportional to this, gives, on the basis of a 70-year length of life in man, the figures shown in table 2. It should be kept in mind that the results were obtained from neutron irradiation. The relative biological effectiveness of neutrons for this effect is not known, but it seems likely, from other data on mutations, that gamma and X-radiation would be less effective than neutrons. It should also be emphasized that the effect observed here is probably a maximum one, since the offspring were obtained from matings made between 19 and 23 days after irradiation. Our data from experiments on mutations at specific loci [13] indicate that the sperm utilized in matings made within this time interval would have been derived from cells in a sensitive stage of gametogenesis at the time of irradiation. From approximately 2 to 4 times as many mutations are recovered from this stage as from the spermatogonial stage, which is the important one so far as radiation hazards in man are concerned.[6] It is also possible that the spectrum of mutations from irradiated spermatogonia would be qualitatively different and, conceivably, less effective in shortening life. However, there is no direct evidence of this, whereas there is evidence from our specific loci studies that some mutations induced in spermatogonia have, even individually, a dominant effect on length of life that is detectable. To summarize this paragraph, it should be remembered that the estimates given in table 2 are based on neutron irradiation of a postspermatogonial and sensitive stage in gametogenesis and that X- or gamma irradiation of spermatogonia would almost certainly produce a smaller effect.

Another way of considering the magnitude of the observed results, so far as its human implications are concerned, is to compare the shortening of life in the offspring of irradiated fathers with that in the irradiated individuals themselves. The data on shortening of life of the males exposed to this same detonation will be presented in detail elsewhere. Briefly, the percentage shortening of life of these animals, based on 24 controls and 128 exposed animals, is 0.078 percent per r. e. p.

The present data, expressed in the same form, give 0.077 percent shortening of life in the offspring for each r. e. p. received by the father; that is, approximately as much effect as on the exposed individuals. Thus the best estimate from our present data is that, for neutron irradiation of the sensitive stages in spermatogenesis, the shortening of life in the offspring of irradiated males will be similar in magnitude to that in the exposed individuals. Again, the effect from irradiation of spermatogonial stages would probably be less.

Whether the *ratio* of effect in offspring to effect in exposed individuals will be different for X- and gamma rays from that observed for neutrons will, of course, depend on whether the relative biological effectiveness of neutrons is different for the effect on the offspring and the effect on the exposed individuals. Present, incomplete data on these points give no grounds for expecting that the ratio of effect in offspring to effect in exposed individuals will be less for X-rays than for neutrons. Weighing the evidence reported here, and making

[12] The Biological Effects of Atomic Radiation: Summary Reports (Washington: National Academy of Sciences, National Research Council, 1956).
[13] W. L. Russell, USAEC Unclassified Report ORNL-2155 (Washington; Office of Technical Services, Department of Commerce, 1956).

some allowance for the many uncertainties, it seems reasonable to predict that, even under the conditions of radiation exposure in man, shortening of life in the offspring of irradiated fathers will be between 10 and 100 percent of the shortening of life in the exposed individuals themselves. It should be remembered that this excludes an additional effect on the offspring; namely as measured in the mouse, death before weaning age. Also, and more important, since the shortening of life is probably the result of mutations with slight dominant effects, the damage would not end with the first-generation offspring but would, to a certain, and probably large, degree, be transmitted to later generations.

Summary.—Length of life in the offspring of male mice exposed to moderate doses of neutron radiation from a nuclear detonation is shortened by 0.61 day for each r. e. p. received by the father over the dose range tested. This figure excludes death before weaning age. The 95-percent confidence limits are 0.14 and 1.07 days per r. e. p. Extrapolating to a proportional shortening of life in man gives 20 days per r. e. p.received by the father as the point estimate and 5 and 35 days as the 95-percent confidence limits. The offspring were obtained from matings made from 19 to 23 days after irradiation and, therefore, represent the effect of irradiation on germ cells in a postpermatogonial and sensitive stage of gametogenesis. It is probable that irradiation of spermatogonia (the stage that is important from the point of view of human hazards) would give a somewhat smaller effect. However, since the present data show an effect on the offspring which is as large as the shortening of life in the exposed individuals themselves, it seems likely that, even when allowance is made for the conditions of human radiation exposure, shortening of life in the immediate descendants will turn out to be of a magnitude that will warrant serious consideration as a genetic hazard in man.

The author gratefully acknowledges the cooperation of Mr. R. L. Corsbie, Dr. E. P. Cronkite, Dr. H. H. Plough, Dr. R. E. Carter, Dr. E. F. Oakberg, Dr. C. W. Sheppard, and Dr. V. P. Bond, all of whom gave valuable assistance in various phases of the work at the test site. The author is also indebted to Dr. A. W. Kimball for statistical advice and computations and to Mrs. Josephine S. Gower and the other members of the Mammalian Genetics and Development Section who assisted with the laboratory work.

Representative HOLIFIELD. Before we have our discussion, we are going to ask Dr. Hardin Jones, to give his presentation.

STATEMENT OF DR. HARDIN JONES, UNIVERSITY OF CALIFORNIA RADIATION LABORATORY*

Dr. JONES. Thank you.

Representative HOLIFIELD. Dr. Jones, this is your prepared statement?

Dr. JONES. Yes.

Representative HOLIFIELD. It will be accepted for the record. Are you going to summarize it?

Dr. JONES. Yes.

(The statement referred to follows:)

STATEMENT OF HARDIN B. JONES, PROFESSOR OF MEDICAL PHYSICS, PHYSIOLOGY; ASSISTANT DIRECTOR, DONNER LABORATORY, UNIVERSITY OF CALIFORNIA

My field is the physiological basis of human health problems. In research I have contributed appreciably to: (1) an understanding of some metabolic disturbances associated with heart and vascular disease; (2) evaluations of the

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cancer problem and especially human cancer therapy; (3) the study of aging in that I have been able to construct a general explanation of aging that can be subjected to experimental study and which has been useful in evaluating factors contributing to improvement or deterioration of health and lifespan. I have been especially interested in estimation of effects of radiation in man.

SUMMARY

In natural radiation exposure and the extent to which it is increased by fallout, we are dealing with effects that are minute compared to other factors of importance to the health of man. Estimation of the magnitude of these small effects depends on determination of the dosages to be expected and the biological responses associated with these doses. This paper deals with the latter factor.

There is no direct evidence that doses as small as these produce any harmful effects; some have therefore jumped to the conclusion that they produce none. There is much evidence that a variety of undesirable effects occurs in cells, tissues, and whole organisms when exposed to doses large enough to establish either a positive or a negative result with reasonable confidence. Some of that evidence is presented here. Since it indicates an effect proportional to dose in the known range, the gap in the unknown range with which we are concerned is filled by arbitrarily assuming that the same proportionality holds. The principal justification for that assumption as a working hypothesis is that to ignore a small factor of risk, if it is real, may be costly if that small risk is applied to a very large population.

The similarity of the effects of all harmful processes, including aging, disease, and irradiation, has been expressed in terms of a concept involving an equivalence between units of the damaging agency and effective increase in physiological age over the chronological age. Factors obtained in this way may be used to estimate the cost in health and lifespan of any of the circumstances tabulated. As a present step, while recognizing the uncertainties of the assumptions on which it is based, the factor for irradiation may be used to compare costs and gains with respect to the atomic energy program. Much more effort should be devoted to the accurate determination of the quantities involved.

COMMENTS TO CONGRESSIONAL COMMITTEE, JUNE 1957

I am trying to evaluate every circumstance which can add up to or subtract from average human health and useful life span. Some of my colleagues jokingly refer to me as a prophet-of-doom because many of my estimates are best explained in measures of morbidity or mortality risks. This is how we must quantify human experience in order to evaluate factors that may truly be worth accepting or avoiding. It is not enough to know something is good or bad for us; we must express this knowledge in quantitative terms in order to be able to compare costs in health or life span with gains in other directions. Very frequently we accept circumstances having a known and measurable risk, such as using the bathtub, crossing the street, overeating, or riding a device for transportation, simply because doing so gives us some definite pleasure or gain that is reasonably more than the estimated cost in risk of harm. However, we are conscious of risk, in that bathtubs now are made with nonslip bottoms, streets have crosswalks and traffic signals, and our mechanical devices are made as safe as is thought to be warranted economically.

Since we are concerned here with the evaluation of radiation effects upon humans, I would like to tell you in some detail about interpretations I believe are reasonable estimates of this problem. I would like to separate information that is generally accepted from that based upon reasonable but debatable arguments; and I shall also use the evident uncertainty regarding answers to critical problems both as a caution concerning current interpretation of results and to indicate needed critical information. My concern for estimation of even minute effects places me at times in awkward situations—when quoted out of context, I may appear to be constructing "scare" stories. This I am not doing. Effects such as I have estimated are too small to be measured directly in individual humans affect estimated numbers of people only when large populations are subjected to the risks in question. Better estimates should eventually be made for each problem we can now discuss, but any estimate will have to depend on statistical studies of large populations.

I state definitely my summary belief that estimated effects of radiation from recommended occupational exposures and from fallout are minute costs compared to the gain to man of abundant useful energy and the extraordinary advantage we have gained for free nations by the awesome presence of effective atomic weapons. I would not, however, assume that, this formula for peace may continue to work indefinitely. Just as defense strategy, economic costs, possibilities for international agreement and other political, economic, and military factors are constantly reevaluated as circumstances change, the biological costs of the atomic age must be reappraised continually.

We all recognize that the world must find more energy to be used for human betterment. Many facts attest to this. Ten percent of the world's population is now using 90 percent of available energy and everyone—whether he already has much or little—wishes to have more of the material comforts made possible through the use of mechanical power. Populations everywhere are increasing, knowledge is increasing, need is increasing. In a world of dwindling available energy, humans very shortly would face a crisis of relative poverty. The availability of atomic energy promises instead a new age for man, marked by extraordinary technical progress that can be the result of both growth of knowledge and increase of efficiency. These asset values are well known; the liabilities are less definitely established. At the moment, we need to find out with much greater precision many basic facts of effects of radiation in man, the biologic costs of radiation exposure, how its effect can be minimized or avoided. It is quite evident that the problems that the atomic age has brought us are more complex than had originally been visualized. At the same time, it is true that the gain through atomic energy is considerably greater than was foreseen even a few years ago. While the physics, chemistry, and engineering of development of atomic energy needed and deserved the great attention they received, it is now evident that the factor of human tolerance of radiation needs further attention because of lack of precise knowledge regarding critical radiation effects. In my opinion, the next stage should emphasize a greater relative effort in this part of the problem, because it is imperative that we reduce speculation and establish estimates of biologic effects with more certainty. At the moment, in spite of the shortcomings of current information concerning these important problems, there seems to be no difficulty in continuing the development of atomic energy and at the same time accepting very cautious limits of radiation exposure.

Much of the information concerning radiation exposure as a problem to man developed almost wholly from the great sponsorship of this field of scientific inquiry by the Atomic Energy Commission. Many of the points currently under critical discussion, such as mutation rate associated with very low radiation doses or the quantitative estimation of fallout, might have been neglected had not those in authority shown a responsible comprehension of these problems and undertaken to support unusual costs as a part of the overhead of development of useful energy.

Now, I would like to present to you an outline of some of the facts and arguments that seem to be of special importance in current estimation of the effects of radiation in man. I wish once again to caution that, in these discussions, we are considering radiation risks that are very much like other commonplace risks to which we have long been exposed, such as driving automobiles—indeed, these newer risks are usually very much smaller.

Everyone wishes that risks to health and life might be reduced to zero. If that could be done, we would all live forever without growing old! Every moment of life, we face certain average risks of mishap—rarely of improvement. These average risks are the sum of many contributing factors, some large, some small. In individual affairs, very small risks are frequently regarded as no risk at all. On the other hand, in a popular presentation of hazards with a view toward emphasizing their importance, any risk that is greater than zero can be presented in a way that attracts concern, even though it may affect only one person in the entire world. A widespread exaggeration of hazards would, of course, have prevented any technical progress. I believe it would be an equally great mistake to dismiss, as being equivalent to zero, those small radiation effects we are now considering. It is true that most of these estimated effects are exceedingly small, and I can give many examples of ordinarily accepted circumstances that modify health to a much greater extent. The problem of differences of opinion derived from the same body of facts, is really the problem of deciding "how small is small," in the light of whatever is to be gained.

Similarity of radiation effect in mammals

In estimating radiation effects in humans, there is a striking similarity to observations established in experimental studies with animals. Cancer induction, radiation sickness, and genetic change occur at equivalent exposures. The average lethal doses are within the same range. Exposures causing burns or tissue damage are comparable. In general, this similarity would be expected from our understanding of the chemistry and physics of radiation effects and from the very similar structure of body cells of mammals of widely varying species. As an approximate truth, damage that is incurred by radiation exposure becomes more apparent as time elapses, so that for many effects, such as the induction of cancer, there is a latent period during which the effect of radiation is rarely observed. Roughly, the latent period for the induction of cancer is relatively long or short, depending upon the relative life span of the species. Thus, greatly simplified, the problem of making comparisons of radiation effect between, say, the mouse and man is a question of the relative time scale. Very similar relations in development of other diseases with time are observed between man and other mammals when time is expressed as a fraction of the life span. In such a comparison, the biology of aging in the mouse is remarkably similar to that of man, if one estimates 1 day's life of the mouse to be equivalent to 1 month's life of man.

Life-span-shortening effects of radiation

The conclusion from all studies of animal populations exposed to radiation in the range of 100–1,200 roentgens of whole-body exposure is that these dosages shorten life span. The shortening of life span of laboratory animals by these exposures is an established fact for either acute of chronic exposure. It is, however, distinctly unproven whether these effects apply on a relative basis to man, though the consistency for the many species of mammals tested lends great plausibility to this hypothesis. If we do make such an assumption, as I have done on several occasions since 1953, one obtains a number that suggests that the average human life span loss at a dose of 100 roentgens may be about 500 days, or about 1⅓ years. We can be certain that some number exists defining radiation induced life-shortening in man, but we cannot state the number with confidence. It can at best only be approximated. It is very important that we do learn what its value is, even though great effort and cost are attached to finding it. In the absence of definite knowledge, we must do the best we can to estimate this quantity.

In discussing this question, several additional considerations arise. Is life span loss proportional to radiation exposure? Most evidence can be interpreted in this sense, but not exclusively. It appears possible that life span loss may be somewhat greater than the above estimate at very large single doses, even though proportionality may hold in the small-to-moderate dose range. For very small chronic exposures, observation of the effect becomes very difficult and it is possible that no effect on life span may exist; however, current data are truly inadequate to test either hypothesis. Nevertheless, in mice, for example, a significantly enhanced and early appearance of tumors has been caused by exposure to 0.1 roentgen per day, the lowest dose yet studied for life span effects; and although no comparison with man can be made on this basis, it may be inferred that some effects occur at low doses. Even though the studies that enable us to speculate upon effects in this dose range have been extraordinary undertakings of technique and labor, we still need to have much more information of this kind to establish the bearing of radiation exposure upon life span loss. One of the most important points concerns whether exposure rate is a factor in the lower dose ranges, since it determines whether one may express this radiation effect in terms of loss per roentgen of radiation exposure. I feel that, with respect to large doses of radiation, the majority of evidence supports a concept that life-span loss is proportional to exposure; and, in the absence of human data on low doses and low rates of accumulation of dosage, it seems safest to assume that the same relation holds at these levels. On this basis, we might say that 1 r. of whole body radiation is perhaps equal to becoming 5 days older. In the absence of definitive information on radiation-induced aging in man, I believe it is reasonable to use this tentative number, even though it may be subject to revision as more certain information is acquired. Study of radiologists and atom bomb survivors may provide a better answer. In this regard there are conflicting opinions and reports. It has been evident for several years that radiologists have 6 to 10 times more leukemia than their expected rates.

Some additional studies of life expectation suggested shortened life span for radiologists. Dr. Shields Warren recently estimated that average age at death was 6 years less for radiologists than for other physicians. Thus, one might infer that radiologists have about 6 years shorter life span. Lewis has recently reviewed this conclusion and reports that, when the distribution of ages among radiologists is considered, the death rate for that group as a whole is no worse—and possibly better—than for physicians in general. However, by using both the individual ages of deaths provided to me by Dr. Warren and the number of registered radiologists, my colleague, Grendon, and I have constructed the age-specific death rates for radiologists. They are found to have the same death rate risk as the general population at ages under 60; but over 60, the death rate is about twice as high as expected. I had come to similar tentative conclusions several years ago by estimating approximate death rates of radiologists from obituary notices in the professional journals. I think it is reasonable to conclude tentatively that radiologists have a higher than expected death risk at older adult ages. This kind of evidence of radiation effect upon man is so important that a relatively great effort should be employed to make a precise study of individuals with known occupational exposures. It is true that the effects are probably very small but they are effects we need to know with relative accuracy, and such study is directly to the joint of estimating the effect of accumulating radiation exposures in humans.

What about dosage measurements in human studies of radiation exposures

Another crucial element in estimation of radiation effect in man is determination of exposure dose. Even though I believe that radiologists may have slightly shortened life span at older ages, there is considerable difficulty in correlating this estimate with a measure of exposure radiologists may have experienced to produce this aging. We simply do not know the average or individual exposures, and direct estimates of exposure can be stated only within rather absurd limits such as 100 r. to 5,000 r. of accumulated whole-body irradiation. There is the possibility, also that only a few greatly exposed individuals may be responsible for the extra deaths, giving a falsely high death rate to the entire group. Such difficulties also plague current attempts to evaluate findings among the Japanese survivors of atomic bombing. It is absolutely necessary to refine estimates of exposure of individuals in order to arrive at a proper evaluation of human life-span effects and to be able to test such data for proportionality or lack of proportionality of induced effects of exposure. A truly valid assessment of radiation effect in man has not been made and would require for its accomplishment the upmost in competent personnel and financial support, on a scale commensurate with its complexity. A great part of the potential and useful evaluation of effects of radiation in Hiroshima and Nagasaki is in jeopardy because too little is known concerning true exposure. The several evaluations of leukemia risk in these Japanese are in doubt because exposure is unknown and evidence for exposure is quite inconsistent with distance from the blast and shielding. Thus, the true magnitude of the leukemia-inducing effect of radiation may be very much smaller or larger than it is now estimated to be. With some effort, the dose might be estimated for accurately for those who developed leukemia and those who were in certain exposure categories. Many critical decisions concerning effects of radiation will need to be made, and it is important to have a proper number to apply in hte construction of these estimates. If such a number can be obtained by making an additional effort, I believe we should do so.

Proportional effects versus threshold effects in man

Attention to effects of radiation first established that there are acute effects, especially marked by tissue destruction, from which recovery subsequently takes place. Recovery in this instance is of the same quality as recovery from any usual kind of injury. As the amount of radiation given in a single dose declines, acute effects decrease more rapidly than the dose, because acute effects are due to tissue destruction and too little damage is done at any one site at doses of about 100 r. and below to evoke measurable systemic responses. Thus, there are some kinds of radiation effect that are not seen at doses of about 100 r. or less. These include immediate radiation sickness, death, burns, ulceration, hair loss, severe anemia, or sterility. All of these symptoms are largely the result of destructive effects of radiation upon rapidly dividing cells. Such cells for the most part are being replaced at a rapid rate, so that the loss of a small number may be quickly compensated. When radiation damage is considered in

terms of acute effects developing soon after radiation exposure, there is general and convincing evidence of at least partial recovery from the effects of exposure. Attention exclusively to this phase of the problem leads some to the opinion that human can tolerate single intense exposures to several hundred roentgens and subsequently recover all of the gross features of normal health.

As we direct our attention to injury of tissues or cells instead of confining it to whether an individual lives or dies, we find in general that effects of radiation are much more nearly proportional to radiation exposure. For whole categories of effects, such as genetic effects, destruction of cells, artifical aging of individuals, and induction of cancer, there is evidence—some completely convincing, some only a reasonable argument—that radiation effects at the cellular level are proportional to exposure to radiation. It is quite possible that some of the effects, such as the induction of cancers or of artificial aging, may not be as likely to occur per roentgen of radiation exposure at low exposures as at higher exposures. These points will be settled only by additional study of the problem experimentally in various animal species and by utilizing every opportunity to study chance human exposures. As a working estimate, it seems reasonable to postulate that effects at low dosage and low rates of dosage accumulation are proportional to know effects at higher doses. First of all, such a working hypothesis seems plausible in the light of the experimental evidence previously mentioned, and secondly, it is a cautious position to take to protect human health.

I believe that the general evidence relating biological effects to exposure shows a remarkable similarity between genetic effects and changes in cell numbers and cell quality in the other body tissues (somatic tissues). Muller, for example, argued as long ago as 1939 that the somatic tissue effects of induced aging and carcinogenic change were quite comparable to genetic effects induced by radiation, and that both effects upon the somatic cells were probably of the same kind as the genetic changes in cells measured directly by the geneticist. Radiation effects on various cells of mammals, such as genetic effects and the survival of cells, including germinal cells, blood-cell-forming cells, and embryo cells, roughly fall into a category of proportional effects of radiation in which the chance of effect is 1 to 3 cells affected out of a thousand, per roentgen exposure.

Radiation effects such as upon growth are quite in keeping with other effects at the cellular level, and human growth change comparable with experimental values of radiation growth suppression in mice. The sum of such evidence suggests that life-span changes and induction of cancers are accelerations of normal aging produced by decreases in cell numbers and cell qualities in amounts proportional to radiation exposure. The effect is frequently made more complicated, it is true, by the ability of the body to repair tissue damage through replacement of injured cells with functional ones; but, fortunately, this modification helps us to escape acute radiation effects.

Were it not for the simultaneous development of atomic energy and broadly supported studies applying to health safety, we would not be in today's position of understanding radiation induction of cancer and other effects to the point of realizing that we should estimate these effects at very low exposures. Only a few years ago, we would have assumed that such effects could not exist. Attention to the effects of radiation in the small-dose range means that these efforts will be extended and that the problem becomes much more difficult technically. It is important to emphasize, in addition to life-span-shortening and cancer-inducing effects, the effect of radiation from generation to generation and upon embryologic development, both of which may be effects that are proportional to radiation exposure into small-dose ranges; if they are, they need to be estimated in terms of human costs.

I would like to turn attention to some of the other environmental effects with which radiation exposure may be compared. For several years, I have been evaluating a large number of environmental circumstances collected from various sources in terms of effects upon the life span and death rate. A summary tabulation is given in tables I and II. The exact numbers attached to some of these effects are in dispute, because there are different choices concerning the estimated magnitude of the effect. Some of the effects are related to reversible circumstances as, for example, marital status, occupational hazards, metabolic disease; other factors, such as radiation exposure, are permanent. The latter class includes traumatic injury, childhood disease, and country of origin. Some of these estimated effects are additive; others are not additive because they are partial measures of the same problem. For example, if one estimates the effect of fat

metabolism on life span from consideration of fat-carrying molecules in the blood, this estimate will already contain the evaluation to life risk that can be obtained from obesity status; and, similarly, the cigarette-smoking effect is also evaluated by blood fats, so that these are not additive. Differences between males and females, urban versus rural dwelling, and national differences appear to be entirely additive.

Differences in life span in this list are given in terms of change of physiologic age. An individual in a category listed as +3 years—as, for example, when a person's mother lived to be 90 while his father lived the average life expectancy—is considered to have the death risk at any adult age of a person 3 years younger chronologically.

Such effects on life span can also be established for, say, automobile driving or employment in industry. The effect corresponding to recommended limits for human radiation exposure (as, for example, 50 roentgens of accumulated exposure in occupations) is very much less than any of these other estimates, perhaps being —0.7 year. The effect of fallout estimated by these methods to be about one one-hundredth of this value. It is quite obvious that each of these effects can be worth our attention, although the average person is not aware at any time that change is taking place. Even such effects as a difference of 10 years in physiologic age are quite unlikely to be noticed by the casual observer watching the tide of life from day to day. Detection of these effects is possible only by employing statistical tools, and the conclusions apply only to averages—not to any one specified individual, except in terms of the statistical concept of risk.

The additive nature of disease categories and factors underlying development of disease suggests a very important point with regard to radiation effects, namely that, even though we cannot reverse the effect of radiation damage itself, it should be possible to counter this effect by enhancing health in other ways. The general recession of disease and improvement of health over this century are clear evidence that great gains can be made in the direction of better health and longer useful life. Some of these gains may more than offset the adverse effects of radiation, and some of these gains may be expected to arise as the direct result of the AEC program in research.

TABLE I.—*Relative displacements of physiologic age by factors that accentuate aging or loss of life span (minus time) or retard aging (plus time)*

REVERSIBLE	Years
Country versus city dwelling [1]	+5.0
Married status versus single, widowed, divorced [1]	+5.0
Overweight: [2]	
25 percent overweight group	−3.6
35 percent overweight group	−4.3
45 percent overweight group	−6.6
55 percent overweight group	−11.4
67 percent overweight group	−15.1
Or an average effect of 1 percent overweight	−.17
Occupational exercise versus sedentary occupation [3]	+5.0
Smoking: [4]	
1 pack cigarettes per day	−7.0
2 packs cigarettes per day	−10.0
Atherosclerosis: [5]	
Fat metabolism from consideration of cholesterol or lipoprotein concentrations in human serum:	
In 25 percentile of population having "ideal" lipoprotein concentrations	+10.0
Having average lipoprotein concentrations	0.0
In 25 percentil of population having elevated lipoproteins	−7.0
In 5 percentile of population having highest elevation of lipoproteins [9]	−15.0
Diabetes: [6]	
Uncontrolled before insulin—1900	−35.0
Controlled with insulin:	
1920 Joslin Clinic record	−20.0
1940 Joslin Clinic record	−15.0
1950 Joslin Clinic record	−10.0
Antibiotics	+

[1] Vital Statistics of Denmark, Netherlands, Sweden.
[2] L. I. Dublin and H. H. Marks, Mortality Among Insured Overweights in Recent Years, presented at 6th annual meeting, Association of Life Insurance Medical Directors (Metropolitan Life Insurance Co., October 1951).
[3] The Registrar General's Decennial Supplement, England and Wales Occupational Mortality, pt. I, 1951 (Her Majesty's Stationery Office, London, 1954).
[4] E. C. Hammond and D. Horn, The Relationship Between Human Smoking Habits and Death Rates, Journal American Medical Association, in press, presented at annual meeting, American Medical Association, New York, June 4, 1957.
[5] J. W. Gofman and H. B. Jones, Obesity, Fat Metabolism, and Cardiovascular Disease, Circulation 5, 514 (1952).

PERMANENT	Years
Female versus male sex [1]	+3.0
Familial constitutions: [7 8]	
2 grandparents lived to age 80	+2.0
4 grandparents lived to age 80	+4.0
Mother lived to age 90	+3.0
Father lived to age 90	+4.4
Both mother and father lived to age 90	+7.4
Mother lived to age 80	+1.5
Father lived to age 80	+2.2
Both mother and father lived to age 80	+3.7
Mother died at 60	−.7
Father died at 60	−1.1
Both mother and father died at 60	−1.8
Recession of childhood and infectious disease over past century in western countries	+15.0
Life insurance impairment study: [7]	
Rheumatic heart disease, evidenced by—	
Heart murmur	−11.0
Heart murmur plus tonsillitis	−18.0
Heart murmur plus strep infection	−13.0
Rapid pulse	−3.5
Phlebitis	−3.5
Varicose veins	−.2
Epilepsy	−20.0
Skull fracture	−2.9
Tuberculosis	−1.8
Nephrectomy	−2.0
Trace of albumin in urine	−5.0
Moderate albumin in urine	−13.5

[6] E. P. Joslin, H. F. Root, P. White, and A. Marble, The Treatment of Diabetes Mellitus, ninth edition (Lea and Febiger, Philadelphia, 1952).
[7] Society of Actuaries, Impairment Study (Peter F. Mallone, Inc., New York, 1951).
[8] As measured in 1900 (Beeton and Pearson). These effects may be measurably less now, as environment is changing to produce greater differences between parents and progeny. Also, in 1900, it was a greater feat than now to live to be 80 or 90.
[9] This 70 percent difference in distribution of lipoproteins, between 25 percent lowest and 5 percent highest, is equivalent to a total of 25 years in relative displacement of physiologic age.

TABLE II.—*Statistical distribution of lifetime shortening by travel and industrial accidents* [1]

[Calculation based on Vital Statistics of 1949, values for adult white males 20 years and older

All accidental deaths	—2.3 years per individual in United States of America.
Travel accidents:	
Accidents involving railways	—0.06 year per individual in United States of America.
Accidents involving ships	—0.04 year per individual in United States of America.
Motor-vehicle accidents involving driver and passengers.	—0.67 year per individual in United States of America.
Assuming only half of population spends appreciable time in automobiles.	—1.3 years per individual at risk.
Pedestrian motor-vehicle accidents	—0.2 year per individual in United States of America.
Assuming this effect largely involves the urban portion of the population.	—0.4 year per individual at risk.
Aircraft accidents	—0.05 year per individual in United States of America.
Assuming that ¼ of the population (actually, probably much less) uses airplanes.	—0.2 year per individual at risk.
Accidents involving industrial machinery	—0.04 year per individual in United States of America.
Assuming only 30 percent of males are employed using industrial machines.	—0.27 year per individual at risk.

[1] These values are based upon numbers of deaths attributed to accidents; the estimates of life span lost are actually perhaps slightly low because survivors who are maimed, and hence have reduced life expectancy, are not included in these estimates.

Dr. JONES. I will deviate from the prepared statement to save time. I can use the blackboard and anyone reading my ad lib remarks can fall back back on the prepared statement if he has difficulty following me.

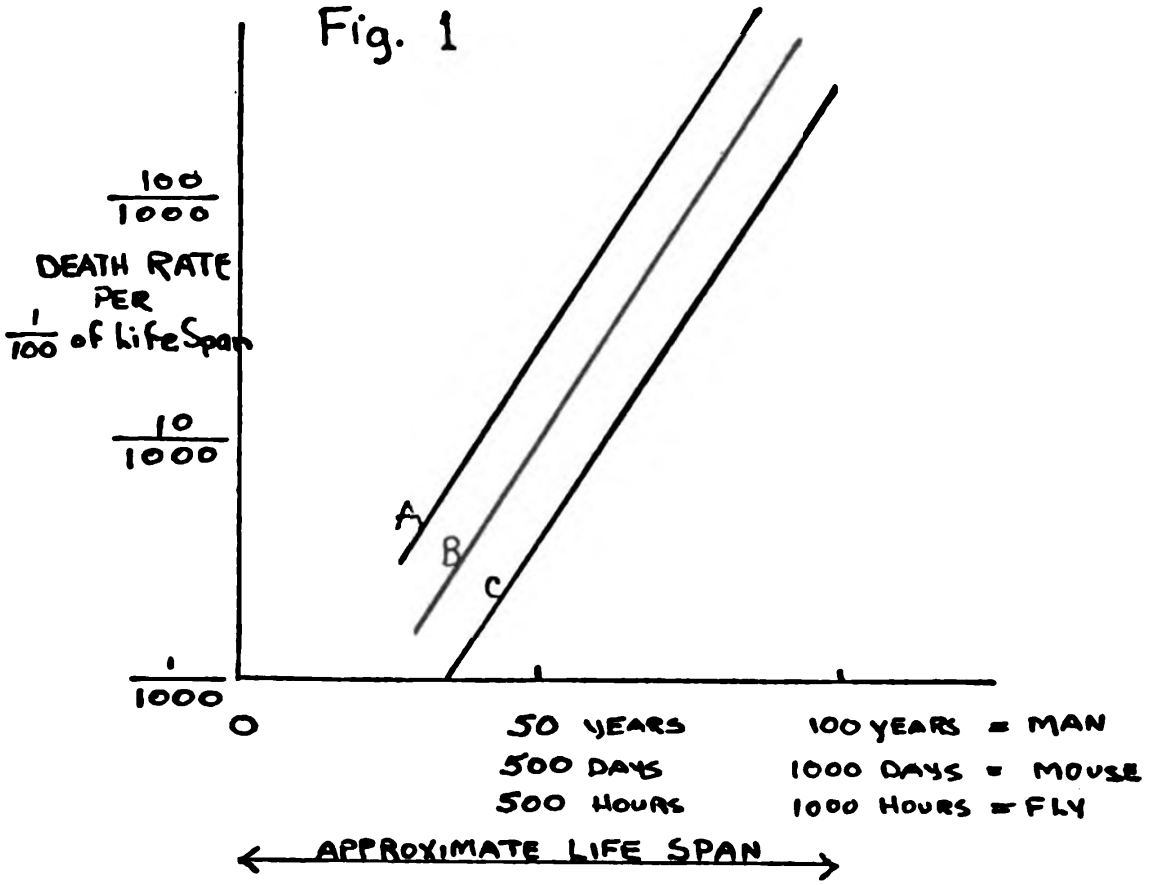
Representative HOLIFIELD. That will be fine.

Dr. JONES. My general problem in health research is to try to evaluate as many things as we can that have some bearing on human biology, human biology from the standpoint of the degree of good health we might achieve and how long we can maintain our life in relatively good health. These things can be expressed in terms of length of life, they also have to do with measures of death rate, because this is how we measure the risks of failing to remain alive.

I would like to present a diagram of this (fig. 1) because I think it helps to understand the problems of human biology. Ordinarily vital statistics for man are presented in units of death rates, that is, deaths per thousand of population per year. This level would be 1 death per thousand per year, this would be 10 deaths per thousand per year, 100 deaths per thousand per year, and so on.

I regret giving information in these terms because they are exceedingly morbid values to deal with. Sometime in the future I would like to turn them around and express them in units of vitality.

We are interested in the problem of vitality and not the problem of morbidity. If we draw this function (line B, fig. 1), this could essentially represent the aging of men or women of the United States. These are approximately the age-specific death rates. If we consider individuals at the age of 30, they have a risk of approximately 2



deaths per thousand individuals per year. At age 50, we find the death risk per year is 10 per thousand and so on.

We find this is a very regular sequence of events. It was described over 130 years ago by Benjamin Gompertz.

The very interesting thing about this is that if the horizontal distance represents life span of man in years, we can convert this equation to the biology of the mouse by changing the time scale to make the life span equal 1,000 days and then our line represents the aging of the mouse. The longer he lives, the larger the risk of dying.

One can also interpret the increasing death risk on the basis that physiologic degeneration is an accumulative affair. The more degeneration accumulates, the more likelihood there is of further decay; and the more decay in toto, the more the death risk. These concepts are relatively important to us because they may explain the very great differences in death rate between populations, either of humans or mice. If we plot the death rates for several populations of mice, we might get marked differences in death rate at each age (lines A and C, fig. 1). The interesting thing is that the biological equation always gives us the same slope. That is, the rate of progression of the death rate tendency is always the same for that species.

In this case, the death rate doubles for each age increase of approximately 80 days for the mouse, or approximately 8½ years for man. Otherwise, the equations are very much the same. The range of variations shown (a factor of 4) in death rate at any age occurs under average circumstances.

I can illustrate some of the differences among human populations. If we say this line represents death rates in the United States today, the Scandinavian countries are in a much better position, with lower death rate risk on the average. We can separate out some population

groups in the United States and find intrinsically low death rates, as good as those of the Scandinavian countries.

What has been happening? If we go back at least 100 years, say, to 1850, we find high death rates; and we find that over the course of the last century, throughout the Western World, death rates have been shifting in the direction toward lower and lower values, even though the rate of change of the death rate with age has remained characteristically the rate of change for man. This helps us to identify many factors that are probably of importance to us in a public health sort of way. I do not think that some of these things can be established with great certainty, but at least the effects of certain conditions have been identified as existing or not, which is quite important to us. One of the main things that has caused a shift from very high death rates in adult life to very low death rates at the same age, has been the recession of the childhood diseases. As childhood diseases have been eliminated, due to progress of nutrition and public health and medicine over the past century, we find that health in adult life has also been better and better, as evidenced by lower death rates.

We can identify a number of factors that have an effect on health. I will put some of them down. The recession of childhood diseases during the last century has added about 15 years of useful adult life. We can tentatively estimate differences associated with exercise—a deplorable thing to discuss when we do not get enough of it. The gain due to exercise might be of the order of 5 years. The effect of obesity, based on life-insurance statistics, is apparently proportional to extent of obesity, where 1 pound of overweightness is equal to 1 month of lifespan.

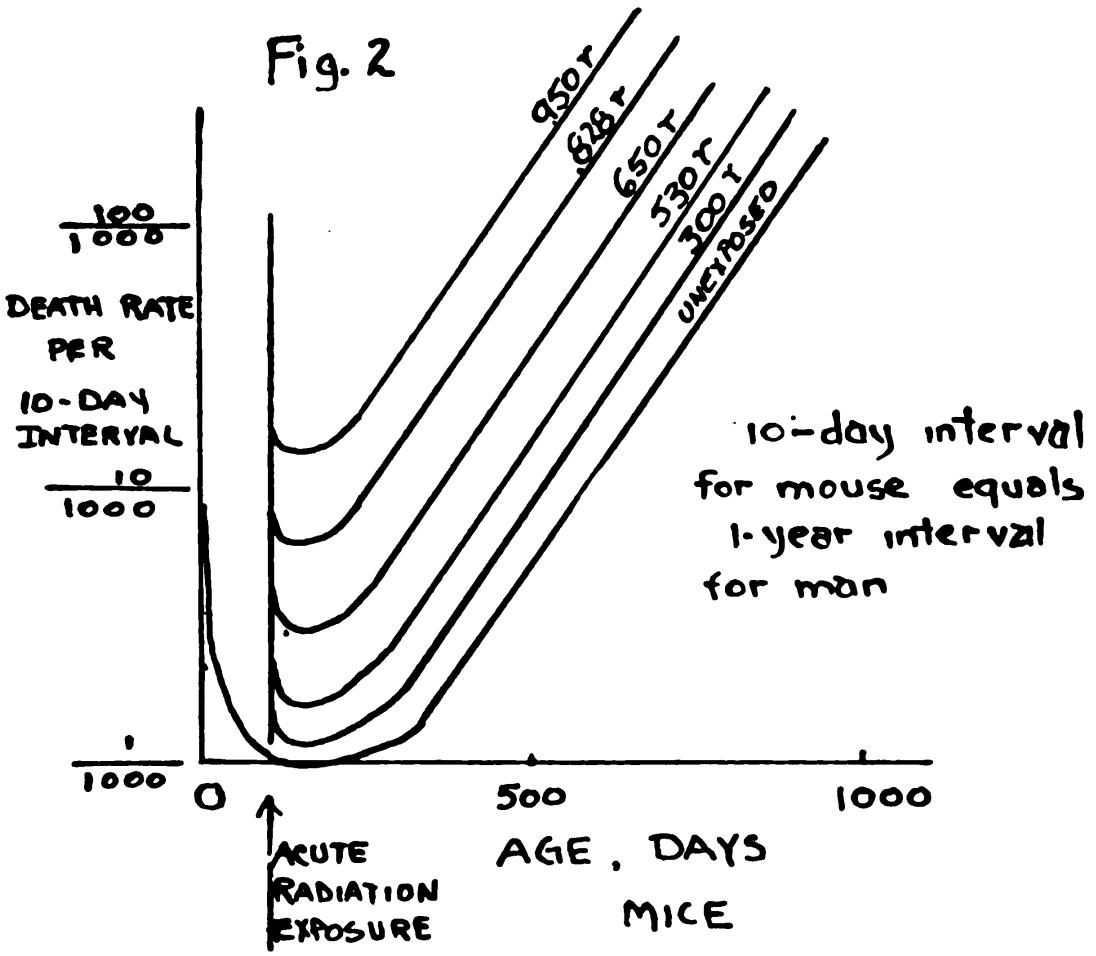
If you think of it the other way, if you are 1 pound leaner, it works in reverse or you have a 1 month gain.

There are differences between living in the country and the city. There are very great differences between various population groups by nation of origin, not related to politics but to human environmental factors.

This type of analysis is important to us because it gives us a background to use with respect to the irradiation problem; particularly, if we look at the effects of irradiation on the mouse, we can use these same equations.. We will be talking in terms of a lifespan of a thousand days instead of the human lifespan of 100 years. The biology of aging of the mouse is like this [drawing on blackboard] (fig. 2). If we give single doses of radiation early in life, what we find is that the death rate goes up momentarily, due to the acute effects, and then, among the mice that recover, there is a displacement of the death rate just as though these mice were already older than they were before.

This apparent aging is the primary thing that characterizes the radiation effect on mammals; the general things that cause death are just about like the things that would have caused death anyway if the animals had been a little older. There are certain things that might really be specific for radiation toxicity, but in general radiation injury represents an increase in natural tendencies toward death. If we give twice as much radiation, this line would move up twice as far.

This brings us, then, to the concept of proportionality. As far as this kind of increase in the death rate is concerned, we seem to have a linear displacement that will move the line toward higher death



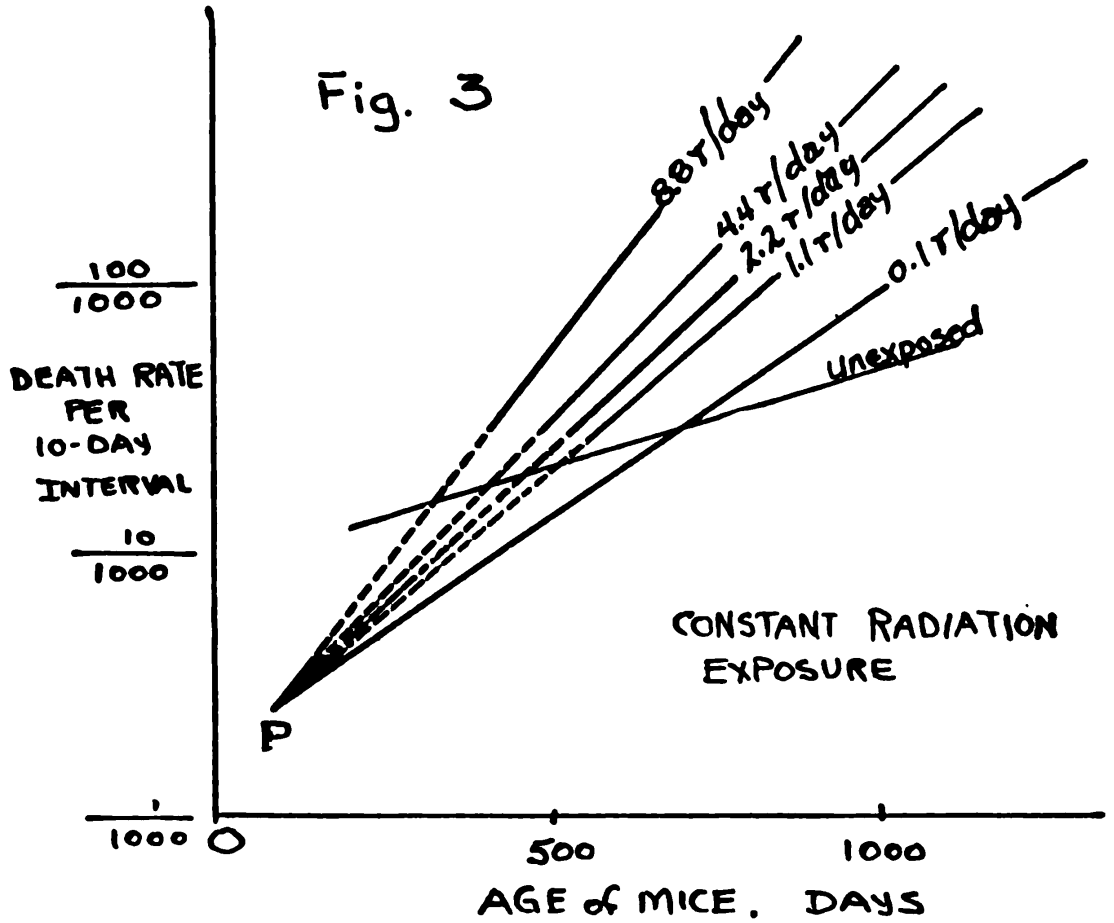
rates in amount proportional to exposure. The amount of injury, which is measured by the increase in death rates, is proportional to the amount of single-dose exposure between 100 and 1,200 roentgens. We can't go to higher exposures because the best techniques we have available will not enable these animals to live beyond the acute phase.

So this gives us one of the best illustrations of proportionality of radiation effect. Even so, there is a great deal of debate among my colleagues as to whether we should really conclude that these effects on life span are still proportional at very low doses. The reason for that is that the basic information we have to go on is relatively scanty. In spite of the fact that we would like to have better data, it has taken a great deal of effort on the part of most of our great laboratories to accumulate the information we now have.

Some of the experiments have been rather great undertakings in terms of numbers of animals, housing of animals, and the numbers of scientific investigators that have been a part of these studies.

A very large study was done in Operation Greenhouse, one of the atomic blasts in the Pacific, where large numbers of mice were used, and where we have good tests of the proportionality. Even in this case there was a difficulty in estimating the physical doses of these animals, and in the higher dose ranges it looked as though the effects on life span were relatively greater than at the lower dose rates. However, at the lower dose rates, the concept of proportionality still seems to hold in that the curve becomes a straight line which seems to go through zero effect at zero dose.

Some of the other difficulties in establishing proportionality of radiation damage are evidenced in terms of another effect that was demonstrated in mice, and that is the chronic radiation exposure ex-



periment of the late Dr. Lorenz. Fairly large numbers of mice were used in this study. In this case, when radiation is given constantly, one does not find that these lines are displaced in a parallel fashion; but, because the little increments of radiation damage are constantly being given, the line has a steeper slope, and the slope of this line will increase in proportion to the amount of radiation given.

So then, if radiation began at this point (point P, fig. 3) and we have varying dosages—maybe this is a tenth of a roentgen per day, this might be 1 roentgen per day, 2, 4, and 8—one can see that the change of death rate with age becomes steeper and steeper as we go to the higher chronic exposures. Unfortunately, with respect to interpretation of the results at the lowest dosages, the control mice were housed separately.

As far as the direct comparison of slope of the death rate curves is concerned, the unirradiated controls seemed to have a lesser slope than even the 0.1-roentgen-per-day group. But when the curves are put together, the control mice intersect in this fashion, (fig. 3), so that early in life the control mice had a higher death rate than the mice irradiated at 0.1 r. per day, and this variation essentially happened twice. So that the direct observation is that the animals that had a tenth of an r. per day lived slightly longer than those that had no irradiation; but the former group did have twice as many tumors as the latter.

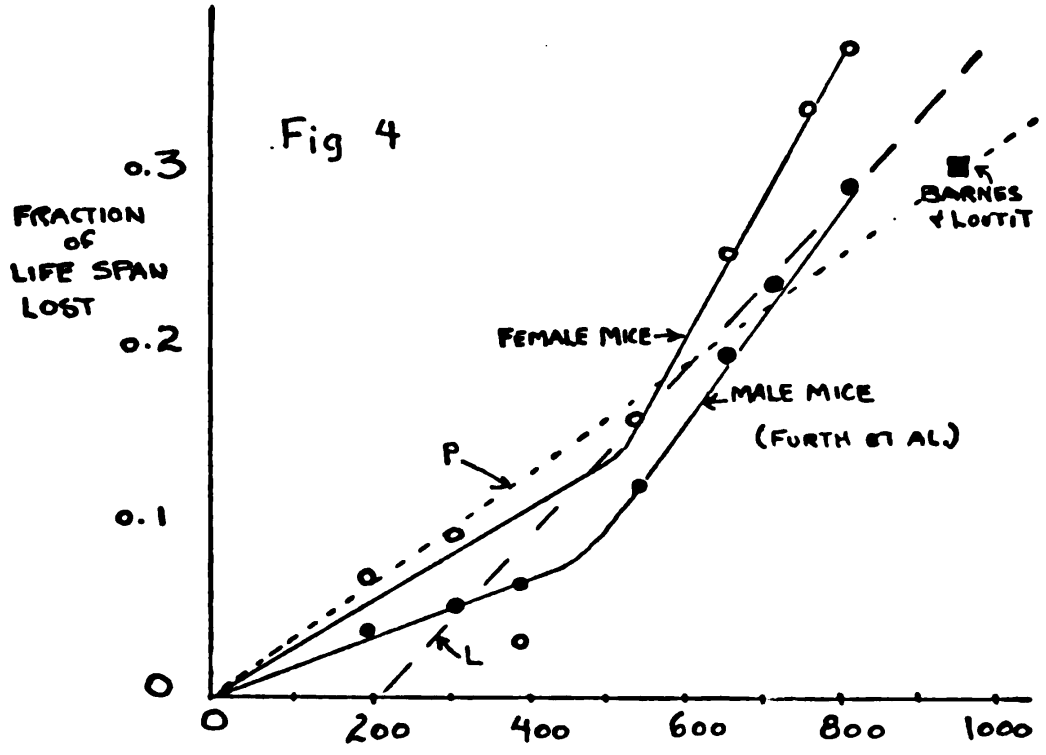
Such differences in death rate are seen quite commonly among animals that are housed separately in slightly different environments, in spite of every effort to make conditions identical. In such cases, however, the slope of the line has remained the same, but the position of the line has been displaced.

I don't know any way out of this particular dilemma except to get further information from repetition of these experiments. The anomaly has given some people a bulwark to fall back upon in arguing that perhaps there is a threshold in terms of radiation effect on the life span. On the other hand, I would place little confidence in this argument, because these animals were housed separately.

Representative HOLIFIELD. What do you mean housed separately and controlled?

Dr. JONES. The experimental mice were chronically irradiated, so they could not be kept in the same room or region of the building as the control mice, which are kept in a normal environment for comparison. They had to be housed in a different room. These particular control animals were even raised at a different time period because the animals that were contemporary with the mice that were irradiated happened to have an infection and the colony was lost. This limits the interpretations one can place upon these experiments.

Even so, expected effects of 0.1 r. per day are so slight, and the numbers of animals are so small, that the results do not let the investigator test significantly either way whether the effects of irradiation on life span are subject to a threshold or are continuous in the small-dose range (fig. 4).



Solid lines are best fit, aging proportional to dose
LINE-P, possible fit, aging proportional to dose
LINE-L, possible fit, aging beginning at a threshold of 200 roentgens

So we have to conclude in general that there is no direct evidence of life span effects in the small-dose range. We can only draw inferences from effects at higher doses. But I believe if we add everything we have available in the higher dose range, of the order of 100 r. up to 1,000 r., that the effects seem to be reasonably proportional to

radiation exposure. Just as the geneticists have trouble in going below 25 roentgens in testing proportionality, I think we have trouble going below 100 r. in establishing that life span effects can occur at very low doses. It is certainly possible to test for radiation effects in this region, but it will be difficult and the testing will not be completed for some time to come.

I also think that everywhere possible we should make an effort to obtain such information directly from man. Of course, there is the study of the Japanese. There is also the study of the radiologists that we discussed briefly yesterday. These studies have some limitations. If one makes the best estimation possible of the death rate for radiologists and compares it with the white male population, it does appear that up to the age 50 the population of radiologists and the white male population have similar death rates. Above this age, the death rate of radiologists is increased above the general population, so that the displacement in death rate, which corresponds to age, makes radiologists effectively at least 10 years older than nonradiologists of the same chronological age. This really only tells us that a life span effect of radiation can occur in man. It is very difficult to say what the ratio of life-shortening to radiation dose is.

It is very difficult to say what the exact number is because the numbers of radiologists have been increasing in each year's time. Over the last 20 years, the number of radiologists has increased by a factor of 3 or 4. With this increase, there has been an increase not only in the younger ages but in all ages. So we have no idea as to what the average dose of radiologists may have been. We can only guess that this 10-year difference in aging is associated with an average dose of somewhere from 200 r. to perhaps as much as 1,000 r. I personally doubt whether this 1,000-r. value taken as an upper limit could really be expected, because I think the effects of 1,000 roentgens of whole body irradiation would be more severe than we see it in the average radiologist.

There is, however, very little doubt that many of the radiologists who are responsible for this higher death rate are really groups that had much greater than average irradiation, and that the main population of radiologists having much less exposure than these few are really subject to a lower death rate.

It is also important at this time to point out that we really need to have a value that we can say represents the effect of radiation on the life span of man. The best thing we can do directly in the absence of such a number, I believe, is to estimate it from the mouse. Unfortunately, we know so much about the biological equations of aging, how diseases develop in both the mouse and man, that we can use this system of simply converting 100 years to 1,000 days to give us this information directly, and use it with whatever confidence limit needs to be applied, remembering that the information is from the mouse.

When we do that, we will find that, taking the mouse data directly, we might conclude that 1 roentgen perhaps—and the "perhaps" is big—is equal to minus 5 days in terms of individual life span. On the average, the individual exposed to 1 roentgen would be like an individual without this amount of radiation who was 5 days older. You see, for all practical purposes within the model as constructed, radiation exposure simulates the ravages of time. So we are justified from the standpoint of the general logic of these events in making this conversion between roentgens of exposure and units of life span loss.

But this is not all. This value, within the confidence limits of the data, might truly range from 1 day to greater than 15 days. We simply don't know. I would advise us all to look at the problem as carefully as possible and use whatever means we can to get such information.

While I cannot be certain where this value may truly lie, the sum of all information I have available to work with would lead me to trust tentatively as a direct average without weighting it for any factor at all this value of 5 days. If we look at the Japanese data in terms of death rate, it is possible that these will fall somewhere within the liimts of 1 day to perhaps greater than minus 15 days per roentgen. We simply do not know yet.

I had occasion to make some very rough estimates some time back and all I could say was that it looked as though there was a life span loss on the Japanese. As we know, there is quite a leukemia rate associated with it, similar to the increase in leukemia rate in radiologists.

Very briefly, I might examine a few factors that are perhaps related to the shortening of life span. They may help to explain radiation effects. I think this particular result of radiation is possibly due to some combination of two effects: the tendencies for the cells to undergo mutations, such as Dr. Muller and others described as somatic mutations, and also the reduction in numbers of functional cells in the animal. If we look at the numbers of functional cells in the animal, or the effects of radiation on mutation rate, we would be looking in effect at a corresponding increment of the death rate. If we look at the cells directly in terms of the response to radiation. for many cells in mammals we will find that there is proportionality in numbers of cells left alive, and number killed per roentgen.

From a lower value of 10 roentgens up to values of 2,000 roentgens in the rat or in the mouse or human blood cell estimates, it appears that, per roentgen in this range, you would have between 0.3 percent and 1 percent of the cells killed. It looks as though the effect of radiation in mammals under a variety of circumstances, for cells like the marrow cells or lymphatic cells, is within this range.

Also, the cells in the developing embryo, which also have a high rate of cell division, have approximately this same range of radiation sensitivity. We can show that the small effect of radiation on the growth of children irradiated before birth in Japan is about the same rate as this, 0.3 percent per roentgen, and this turns out also to be equivalent to the data that Dr. Russell described a moment ago on his irradiated mice. So we have some general idea that in the various mammals we can study, the effect upon cell numbers seems to be relatively the same among the various mammalian species, and also the life span effects seem to be roughly the same as far as we can test; but it does leave us with the great desire to know much more about these effects, and also to know what kind of number we should estimate for the life-subtracting effect of radiation in man.

Senator HICKENLOOPER. Do I understand that range you put on the board a moment ago from 10 roentgens to 2,000 roentgens, that an exposure of 10 roentgens would kill 0.3 percent of the cells and exposure of 2,000 roentgens would kill 1 percent?

Dr. JONES. The range of 0.3 percent to 1 percent of cells killed per roentgen applies throughout the range from 10 roentgens to 2,000 roentgens. One has to interpret the proportionality in what we call

the hit equation. You cannot kill the organism twice. So that in upper doses very few cells are left untouched.

Senator HICKENLOOPER. That is the thing that confuses on that statement. I would interpret what I understand you to mean that as compared to an exposure of 10 roentgens and 2,000 roentgens that the 2,000 roentgens would kill only about 3 times as many cells percentagewise as the 10 roentgens. That is my understanding of your statement.

Dr. JONES. No. If we test for this range where we have available data in the mouse, regardless of whether we give 10 roentgens or give 2,000 roentgens, we will find that we have 0.3 percent of the cells affected per roentgen. So that, per roentgen, as far as the effect on rapidly dividing cells is concerned, it will be three cells of each thousand cells affected.

Senator HICKENLOOPER. I understand. I missed the equation to the single roentgen.

Dr. JONES. Yes. The remarkable thing is that this is such a good proportionality effect.

Representative HOLIFIELD. Dr. Jones, we want to adjourn at 5 because the committee has to go into executive session on another matter.

Dr. JONES. I can summarize this section. I think life-span effects do exist. I have no reason to doubt this at all. I have some reason to believe that we should look with caution on the argument that a threshold effect exists, although we cannot be absolutely certain that a threshold effect might not exist. But as far as my opinion is concerned, on the basis of having examined all the facts at my disposal, I do not believe a threshold effect is very likely to exist, I think we should try to get better information, not only for this point, but also to find out directly in man how much each unit of radiation subtracts from life span, because in an atomic age everyone is going to need to know this number with a great deal of certainty. At least, it should be determined to 1 or 2 significant figures and not merely within the range of perhaps a factor of 10.

Representative HOLIFIELD. Thank you. Your summary is very interesting, and the statement that you have submitted will be studied carefully.

I have two papers by Dr. Jones that I would like to place in the record at this point.

(The material referred to follows:)

A SUMMARY AND EVALUATION OF THE PROBLEM WITH REFERENCE TO HUMANS OF RADIOACTIVE FALLOUT FROM NUCLEAR DETONATIONS

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ABSTRACT

The tolerable amount of radiation exposure to humans is probably less than formerly estimated. It is shown, however, that accumulated effects of the low-level worldwide exposure to radiation from fallout to date is relatively small. The genetic effects are not large enough to be statistically detectable. The health effects, as expressed in life expectancy, are much smaller than those of such factors as infectious or chronic disease, metabolic disturbances, smoking, obesity, lack of exercise, and environment and marital status. Predictions of strontium 90 levels to be expected in the next 2 decades indicates, however, that bone irradiation may become detectably harmful.

[1] With suggestions and critical review gratefully acknowledged to R. Lowry Dobson, John W. Gofman, John H. Lawrence, Burton J. Moyer, William Siri, Curt Stern, and Edward Teller.

INTRODUCTION

Nuclear detonations form radioactive isotopes in quantities so enormous that they must be recokoned in terms of many thousands of curies, the equivalent of many pounds of the element radium. A portion of this radioactivity is dispersed into the atmosphere and subsequently falls upon the land and sea. However, the vastness of the land, air, and water of the earth provides a means of dilution so great that even these large quantities of radioactive materials are soon reduced by distance and time to exceedingly small concentrations of radioactivity. The problem of radioactive fallout in relation to human beings involves the need to know the quantity that becomes a part of human environment, and to know the effect upon man of ionizing radiation from fallout.

This summary concerns low-level worldwide fallout. It must be recognized, however, that very intense fallout may be experienced in the vicinity of an atomic detonation. For example:[1]

"On March 1, 1954, an experimental theromonuclear device was exploded at the United States Atomic Energy Commission's Eniwetok Proving Grounds in the Marshall Islands. Following the detonation, unexpected changes in the wind structure deposited radioactive materials on inhabited atolls and on ships of Joint Task Force 7, which was conducting the tests. Radiation surveys of the areas revealed injurious radiation levels; therefore, evacuation was ordered, and was carried out as quickly as possible with the facilities available to the task force.

"Although the calculated accumulated doses to the exposed human beings were believed to be well below levels that would produce serious injury or any mortality (267 Marshallese received 14 r. to 175 r.) * * * All of the exposed individuals have recovered from the immediate effects (burns, loss of hair, anemia) without serious sequelæ. Nevertheless it is planned to evaluate the medical and genetic status of the group at appropriate intervals with a view to learning what if any of the known late effects of radiation exposure may be observed. Obviously and indeed fortunately the number of persons (92 Marshallese) receiving 75 roentgens exposure and greater is too small to make it possible to determine with any degree of accuracy the effect on life span."

EFFECTS OF RADIATION EXPOSURE IN HUMANS

The reasonable and superficial evaluation of radiation hazard is that humans can obviously tolerate exposure to several hundred roentgens, recover from immediate effects, and remain in "normal" health and functional capacity. Recently, however, we have become aware of deleterious long-term effects of radiation which, however subtle, appear to be proportional to the total quantity of radiation exposure and may be assumed to act even at very low levels of irradiation. Except for these long-term changes, our understanding of radiation effect usually has been concerned with two important facts that dominated our thinking about these problems:

(*a*) For certain kinds of radiation damage and injury, there is recovery. Individuals recover from acute symptoms produced by sublethal radiation exposures even though they may show general sickness, burns, loss of hair, anemia, etc. Recovery from acute radiation effects is analogous to recovery from any other acute injury or infectious process in which damaged tissue is healed and repaired.

(*b*) These obvious signs of radiation effect are associated with relatively large single doses of radiation (greater than 100 r.). As dose size is decreased, detectable acute effects decline, becoming disproportionately small, so that there is a true threshold of dose of irradiation, at about 100 r., below which these particular acute manifestations of radiation do not occur.

THE PROPORTIONAL-EFFECT CONCEPT OF IRRADIATION

Recent evidence that long-range effects of radiation simulate aging effects comes from a variety of sources and is consistent with information relating radiation effects with genetic change and changes in cell-population numbers and quality. Evidence and logic support an argument that small increments of radiation-induced morbidity persist as small permanent changes in body functional struc-

[1] Quoted from Charles Dunham, A Report on the Marshallese and Americans Accidentally Exposed to Radiation From Fallout and a Discussion of Radiation Injury in the Human Being (United States Atomic Energy Commission, July 1956).

tures, which become detectable as aging, neoplastic disease, and genetic change (see appendix A). However, it remains to be proven experimentally that these effects do occur as the result of small irradiation exposures. The testing of this question is not likely because it would involve great technical difficulties. Attempts to procure some evaluation of the problem relating small-doze (5 r. to 10 r.) effect to life span would involve study under uniform conditions of perhaps several million mice. It is pointed out that in order to establish the effect of the smallest doses as yet measured for genetic effect, namely 25 and 50 r., the geneticist Curt Stern and his associates worked for 6 years and examined approximately 50 million individual flies. They came to the acceptable conclusion that these small exposures have the same effect per roentgen upon gene mutation as at higher exposures to radiation. Ionizing-radiation effect, in the depression of blood-forming function and blood-forming cells, is proportional to radiation dose even down to 5 r. (Hennessy and Huff). Other effects upon blood cells, leading to abnormal doubling of the cell nucleus, are now reasonably established by Dobson in the range of 0.1 to 0.3 r. of single exposure, but are not yet tested for proportionality.

There is no reason to doubt the general evidence of a proportional effect of radiation; but it is also possible that linear extrapolations of higher doses to the small-dose range may not give a true representation of the problem. It is known for some kinds of cellular response to radiation that there can be no effective change in function until two or more similar critical entities within the cell are affected. Some kinds of observed injury, however, appear to depend upon the effect on one critical entity per cell; and other observed injuries may be the result of damage to any one of a number of critical functional parts. The response that depends upon a chain of two or more detrimental changes shows a lesser apparent effect in proportion to radiation exposure at the low-dose ranges. Although this kind of irradiation response does not argue against extrapolation of radiation effect, it may explain a factor of —2 or —4 buffering against detectable radiation effect in the lowest exposure ranges; or it may even have the opposite effect, because radiation effect can add onto partially initiated dysfunctional changes in structures that otherwise would have remained functional.

Radiation effect is most frequently estimated in animals that are rather uniformly irradiated over the whole body. Thus, we are usually generalizing from the observed result of whole-body exposure. In some studies (Kaplan), shielding a relatively small portion of the bone marrow from radiation may protect the animal from generation of thymic tumors. In others, local irradiation is associated with induction of cancer in that region, quite independently of exposure or shielding of the remainder of the body.

The problem of estimating radiation effect and making recommendations concerning it is not the simple problem of avoiding exposure at levels at which there is a detectable or predictable response. This is especially true when considering radiation effect through systems that allow proportional extrapolation to very small radiation exposures. It is always important to keep radiation exposure to a minimum; but it is also important in the understanding and evaluation of the relative importance of radiation effect to establish its place in the entire climate of factors that can modify health. Similar—and, at times, greater—effects upon health can be shown to result from a large number of common environmental factors.

Also, the problem is not simply that of effect of body irradiation upon health. It is necessary to evaluate the effect upon human beings of all known phenomena resulting from the onset of the atomic age, including general socio-economic factors related to our well-being, which are dependent upon progress and the development of useful energy.

ADVANTAGES MINUS COST EQUALS NET GAIN

The sum of evidence would lead to the conclusion that radiation probably does affect man's health subtly, and—like money and time—it should be exchanged for equivalent advantages.

Since the usefulness of atomic energy—including material and energy gain and defense measures of prime importance—is a positive result, and the radiation effect upon humans generally is a negative result of the atomic age, atomic-energy usefulness minus harmful radiation effect must be equated to the net gain. Therefore it is critically important to estimate hazard quantitatively, and to be mindful of other factors while doing so. However, there is no unanimity of opinion at this time as to the precise balance that should be achieved between advantages and disadvantages of use of atomic energy, because certain

qualifying factors are still too poorly known. Uncertainties exist which can mean either underestimation or overestimation of the effect of radiation. This brief synopsis roughly appraises the biological costs of exposure to radiation and presents information which must largely guide decisions in the interim until more precise information on radiation effect is available.

A summary of current knowledge of radiation tolerance or hazard and fallout is provided in several major public documents that have appeared in 1956 in broad survey of the problems to man of atomic radiation and fallout.[3,4,5,6,7]

THE CONCEPT OF MAXIMUM PERMISSIBLE DOSE

Early estimates of that amount of exposure to ionizing radiation which constitutes a permissible occupational hazard placed the upper limit at 0.1 r. per day. Such a value was exceedingly conservative in view of information available at the time it was established. It is lower by a factor of 50 than chronic exposures leading to physiological disturbances and radiation sickness, and by a factor of 1,000 to 5,000 than the dose which, in a single exposure, might threaten life. Also, at the time it was proposed, 0.1 r. was the lower limit of radiation exposure dose known to elicit any biological response. Evidence on the magnitude of physiologic response of the individual to radiation in the range of a few hundred roentgens has not changed: but extensive information on effects of lower levels of radiation has recently appeared. This knowledge requires a reevaluation of the cost to humans of radiation exposure in terms of (*a*) genetic effects, (*b*) shortening of lifespan, (*c*) induction of cancers, (*d*) destruction of tissue, (*e*) congenital malformation, and (*f*) effects upon young individuals. All these effects appear to be proportional to the exposure to radiation, and have been largely responsible for a recent downward revision in maximum allowable exposure to radiation.

THE GENETIC EVALUATION OF RADIATION EFFECT

Up to 1946 estimations of the genetic effects of radiation had placed the quantity necessary to double the mutation rate per generation in the fruitfly at about 50 r. (Muller, Stern), but with some uncertainty, so that the true value might have been 80 r. or 35 r. At that time, there had been relatively little comprehensive evaluation of the range of genetic sensitivity to radiation in mammals or man. At the present time, the mutation rate per generation for the fruitfly is known to be doubled over the natural rate by about 50 r. (Stern). Through genetic study of irradiated mice (Russell), the amount required to double mutation rate per generation in the mouse is partially established also at approximately 35 to 80 r. Wright has estimated from evidence now available that the mammalian mutation rate may be doubled by as little as 3 r. or as much as 300 r. The best current estimates place the mammalian mutation-doubling dose of radiation at about 50 r. (4).

As an approximation, each species appears to form in natural circumstances about one new mutation in a generation time. The fruitfly lives a short time in about the same radiation environment (estimated roughly at 0.1 r. per year) as man. In its life span of 20 or 30 days, it can accumulate only the minute quantity of 0.008 r. Thus, if 50 r. in the fly produces an additional number of mutations equal to those which occur naturally, radiation can account for only a part of the natural mutation frequency, namely, the fraction

$$\frac{0.008}{50}=\frac{1}{6{,}000}.$$

Hence, at background radiation, only 1 observed mutant in 6,000 is suspect of being induced by radiation. In humans, the life span up to average reproduction age is about 30 years, lived in the same environment of 0.1 r. per year, or a total of about 3 r. by average reproduction age. Thus, if 50 r. is estimated to double the human mutation rate, radiation from natural sources may be expected to account for

$$\frac{3}{50}=\frac{1}{17},$$

[3] National Academy of Sciences, The Biological Effects of Atomic Radiation—Summary report, 1956.
[4] National Academy of Sciences, The Biological Effects of Atomic Radiation—Report to the Public, 1956.
[5] British report, Radiation Hazards to Man, Cmd 9780.
[6] Willard F. Libby, Current Research Findings on Radioactive Fallout, Proc. National Academy, December 1956.
[7] M. Eisenbud and J. H. Harley, Radioactive Fallout Through September 1955, Science 124, 8215 (Aug. 10, 1956).

or approximately 6 percent of the naturally occurring mutations. If we accept the lowest possible value of 3 r. for the mutation-doubling dose, we would have as the fraction attributable to radiation

$$\frac{3}{3}=1,$$

and radiation could account for the entirety of mutation changes in humans.

The fallout of radioactive materials through 1956 has increased the radiation exposure of gonadal tissue by an amount estimated as approximately 0.004 r. per year (see table V-D) (largely from ingested cesium-137 (4) and deposits on the ground (6)). This is an increase of approximately 3 percent over natural radiation exposure.

The recommended limits of radiation exposure in man will be affected by information on the quantitative relationship between ionization and mutation and the understanding of the natural mutation burden. Should we estimate the level of radiation likely to double the natural mutation frequency in man as 25 r. or 3 r., we will be at least 2 to 20 times as concerned about the genetic problems associated with radiation exposure as we are under the current assumption that the human mutation rate is doubled to 50 r.

Genetic studies of irradiated Japanese have been carried out by the Atomic Bomb Casualty Commission at Hiroshima and Nagasaki. A 10-year study has been analyzed by S. V. Neel and W. S. Schull.[8] The principal result is that no measurable increase in mutation rates was observed. They measured biological characteristics that could reflect genetic state and genetic change, such as stillbirths, male-female birth ratios, and congenital malformations. The results of all observations of this kind can be interpreted either as demonstrating no measurable increase in these events, which are associated with mutations, or as showing that, had the true congenital malformation rate been doubled, there would be only 90 percent probability of discovering even this increase. Thus a small increase in congenital evidence of genetic change would not have been detected.

The results of the study of the Japanese indicate that the human genetic effect of radiation is acceptably consistent with the range of response estimated from mammalian genetic experiments; and it establishes with certainty that there are no catastrophic genetic effects at low to medium range of radiation exposure in human beings, although catastrophic effects are predicted at high levels of accumulated radiation exposure to whole populations. Many new mutations were probably produced in the Japanese exposed to the atomic bombs; but many of these may have been unobserved because of early lethality, and the rest are overwhelmingly diluted by the vast number of normal genes. This dilution was expected; and the statistical odds are known to be very greatly against the appearance of unfavorable and detectable combinations of mutant genes in any one generation of offspring.

Genetic change is, of course, basic to the concept of the Darwin principle of evolution. For this reason, it is possible that some increase in the mutation rate might be to human advantage in the long run by providing a greater pool of variance from which selection could take place, to our final advantage some thousands of years from now. Some brief speculation regarding the extreme limits of variation may be offered:

(*a*) Humans and other long-lived animals have, as a corollary of their longevity, a less frequent natural mutation rate per gene per unit of time than short-lived species. As an approximate rule, each species appears to have about the same mutation rate per generation time. Thus, it appears that species are in some balance between generation time (or lifespan) and stability of genetic structure.

(*b*) Testing biological capacity for survival under circumstances that increase genetic variation is possible only with species having relatively short generation times. They have a common feature of a potentially great ratio of progeny per parent. These species can therefore survive even if a relatively high proportion of conceptions are incapable of survival and reproduction. Humans in natural selection are at some disadvantage in comparison with species producing a large number of offspring per generation, such as the mouse or the fly. Thus, human

[8] Reported at the First International Congress on Human Genetics, in Copenhagen in August 1956.

genetic tolerance should not be judged from effects of radiation exposures on these more fertile populations.

(*c*) For survival of a species, the ratio

$$\frac{\text{Reproducing offspring}}{\text{Individual}} = \text{must exceed 1.}$$

In humans, lowering of infectious disease toll has brought this ratio to approximately 2. As a consequence, the human population is now doubling in numbers approximately every 40 years. Thus, humans have already achieved some protective reserve against genetic changes toward lower fertility.

(*d*) On a scale of catastrophic genetic misfortune, humans also have the protection of vast numbers of individuals. There are 2.6 billion inhabitants of the world. While this number is small compared with numbers of insects and small mammals, it is still a very large number compared with that of any previous age in the history of man. Radiation exposure, as a cause of genetic change and increase of genetic variance, would be expected to produce that change in a random way. Thus, even with a large increase in genetic variance induced by radiation exposure, if population numbers are sufficiently great, some individuals will remain relatively unchanged. If these individuals were favored in selection, they might replace the less fit, less fertile fraction of the population. Thus, if survival of mankind were the only consideration, population numbers might, through reduction and segregation, achieve selective retention of adequately functional humans.

Several approaches to evaluation of extreme tolerance of human populations to radiation exposure with respect to health and genetic constitution are presented in appendix B. These methods of estimation are difficult and speculative; but they indicate that an additional 2 r./per year (or 50 r. per generation time) of chronic radiation exposure to the average individual in the human population would eventually cancel health and lifespan gains we have achieved recently. Such estimations of impairment of health and estimations of the cost of increasing the genetic variance suggest that human population cannot afford the biological cost of this intensity of chronic radiation exposure, and that there should be extreme caution at this time against increasing the radiation exposure to all people by 10 times over its natural level.

Evolutionary benefits?

It seems possible that human evolution is occurring in some optimal balance between mutation tendency and genetic stability. Fertility, length of life, death rate, and individual usefulness may be highly affected by the number of accumulated new genes,[9] which both add to favorable evolutionary drift in average human vigor and add to the pool of undesirable genes to be selected against. At low radiation levels, such as 10 percent or 1 percent above the natural radiation background (the range of fallout effect), it seems unlikely that long-range genetic disturbances can become an appreciable problem, since the natural radiation background appears to account for only 10 percent of the change in genetic structure per generation. One may speculate further that, in the long run, man may be beneficially affected by good genes yet to be formed, so that increasing radiation exposure and the mutation rate may operate to human advantage. Such an argument is unlikely to convince men who understand some of the dangers of too great a burden of undesirable mutants; it is analogous to an attempt to convince the experienced cook that the baking of her prize cake would be accomplished in half the time at higher oven temperature.

Penrose has evidence of indirect beneficial effect of some recessive lethal genes, which appear to enhance the effect of the functioning gene with which they are matched in individual combinations. This effect is one in which mutation may beneficially add some variance to genetic functional characteristics. On the whole, however, there is a strikingly large mass of information indicating that any genes that can disturb function should be kept to an absolute minimum.

Unfortunately, there are still many unanswered questions facing geneticists on the topic, "What is the effect of undesirable genetic burden on the quality of humans?" Fully satisfactory experimental measures have yet to be applied to this problem. One approach that has led to considerable speculation is through

[9] Transformed genes are, with rare exception, nonfunctional, lethal, or undesirable.

estimations of the numbers of undesirable mutations carried by the average person. Estimations of this burden place it within the small range of 5 to 15 undesirable genes per average individual[10] (4). This value is the equilibrium resulting from approximately one such gene gained and one lost in each generation.[11] Thus it has been pointed out that if, through increase in radiation exposure, the genetic gain of undesirable genes increased from 1 per generation to 2 per generation, there would be a relatively great reduction in the quality of the best 25 percent of individuals (assuming that reduction in quality of offspring is proportional to the number of undesirable mutations per individual). Because of speculative—but reasonable and cautious—arguments of this nature, geneticists have uniformly cautioned against allowing any major proportion of the population to accumulate radiation as high as 50 r., which is the amount estimated to double the human mutation rate.

LIFE-SHORTENING EFFECTS

Life-shortening effects of radiation have been observed under a variety of experimental conditions. An experiment of particular significance because of the large numbers of animals and the range of exposure was the exposure of mice to nuclear detonation at "Operation Greenhouse" (Furth et al.). The fraction of life span lost per unit of radiation exposure appears to be essentially the same for a number of species, including the mouse, the rat, the guinea pig, the rabbit, and man. The largest number of experimental observations concerns the mouse. In the mouse, the fraction of the life span lost per unit of whole-body radiation exposure is acceptably constant over a wide range of variation in radiation exposure. The tentative conclusion is that radiation effect simulates aging itself, and that a unit of radiation exposure, regardless of the intensity and duration of exposure, produces approximately the same relative disturbance to body structure in adults of all mammalian species. On the human life span scale, these effects of radiation summarized from small-animal data suggest that 1 r. of radiation exposure is equivalent to 5 to 15 days of physiologic aging. This prediction is confirmed directly in man (with reasonable technical reservations) by Dr. Shields Warren's recent investigation of life span of radiologists compared with physicians not using radiation in their practice of medicine. The average age at death is approximately 6 years less for radiologists than for physicians in general practice or for pathologists, both selected as being relatively unexposed to radiation. The estimation of accumulated radiation exposure in radiologists is uncertain, but has been approximated as 300 to 500 r. Thus, the life span loss, if attributed to radiation, is

$$\frac{-6 \text{ years} \times 365 \text{ days/year}}{300 \text{ r to } 500 \text{ r}} = -7 \text{ to } -4 \text{ days per r of whole-body exposure}$$

Such a number is still subject to considerable possible revision; but many different estimates give values of 1 to 30 days lost per roentgen of radiation exposure, and the probable value for humans is in the range of 5 to 10 days lost per roentgen.

A question exists whether we can justifiably extrapolate effects such as life-span lost per roentgen from measures that are mostly determined in the range of 100 to 1,000 r. The evidence is that, over the range that can be tested, the effect is linearly proportional to the radiation exposure; and the information fits an extrapolation to zero shortening of life span at zero artificial radiation exposure. There is additional evidence in the effects of radiation upon cells (as distinguished from entire organisms), in which lethal damage to cells per roentgen also appears to be proportional to total radiation exposure. Such estimates agree for cells in the mouse, the rat, the rabbit, the guinea pig, and man. This

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[10] Some individuals may have none. The fraction having none or very few diminishes steeply with increasing average numbers of undesirable mutations. Thus, doubling the burden of mutations may reduce the numbers of individuals having desirable genetic combinations to rare events.

[11] The average mutation frequency of 1.5 spontaneous mutations of human genes per generation, as summarized by Penrose, corresponds to 30 mutations per million genes per generation, assuming that humans have about 50 ;000 genes :

$$\frac{30 \text{ mutations per generation} \times 50{,}000 \text{ genes per individual}}{1{,}000{,}000 \text{ genes}} = 1.5 \text{ mutations per individual per generation.}$$

The average mutation rate may be less than this estimate, since one may suspect that the genes usually observed to mutate are perhaps 10 times as mutable as the average gene.

experimental evidence that effect of radiation on cells is in linear proportion to radiation exposure of from 15 to several thousand roentgens provides a reasonable basis for understanding the life-shortening effects of radiation.

Furthermore, the life-shortening effects are consistent in order of magnitude with the genetic effects of radiation upon cells (2 to 3 cells affected per 1,000 cells per roentgen). The genetic effect of radiation has been shown to be acceptably proportional to radiation exposure from 25 r to 8,000 r.

The sum of systematic evaluations of such effects of radiation as mutation induction, cell destruction, and life-span shortening indicates that these effects are permanent and represent the quantum interactions of radiation randomly affecting body cellular structure. The concept of quantum interactions with matter justifies extrapolation to the probability that a single quantum of radiation reacts with an individual molecule.

Although all recent evidence suggests that radiation effects is proportional to radiation exposure, such effects must be viewed together with other common environmental factors that modify health. A scheme is used here in which the effects upon health is expressed as an induction of aging (this is expressed as loss in physiologic lifetime, or minus time, written "$-n$ years") or as a postponement of aging (expressed conversely as lifetime gained, or plus time, "$+n$ years").[12] These factors all appear to have a general action upon disease tendency, and the effect is about the same at any adult age. The list of relative displacements of physiologic age (table I) is given for factors that accentuate aging or loss of life span (expressed as minus time) or retard aging (expressed as plus time). These measures are derived directly from human records. They are grouped according to whether they appear to be reversible or permanent. Most of the effects that are not partial measures of the same state are apparently additive, in the few instances that can be tested for this property. (See p. 1107.)

Certain of these circumstances that modify health are partially interrelated, others may be independent of one another. Estimates of effect upon physiologic age may be additive, depending upon the extent to which they are independent. Thus country against city dwelling may be suspected to include the factor estimated as exercise benefit. The lipoprotein test already contains information that can be estimated partially by relative overweightness, and the lipoprotein tests already accounts for a portion of the smoking effect. Familiar inheritance is independently estimated from each ancestor; male against female differences are equally added to city against country effects, and presumably each separate disease sign in the impairment study is additive.

In further support of the additive nature of effects upon health, each morbidity circumstance that can be quantitatively estimated produces an effect proportional to the intensity of the circumstance. Examples of proportional change in mortality risk with morbidity severity are:

(*a*) Overweight: —0.17 year for each percent overweight

(*b*) Smoking: —0.45 year per cigarette used per day

(*c*) Radiation: —5 to 10 —days per r. of whole body radiation
3 cells killed per 1,000 cells per r. (marrow and lymphatic tissue)
4 cells with chromosome breaks per 10,000 cells per r.
1.4 percent increase in lukemia per r.

(*d*) Atherosclerosis, diabetes, nephritis: End effects are proportional to severity of metabolic error

(*e*) Accidents are proportional to exposure risks

A somewhat similar tabulation can be made of an estimation of the cost of industrial and transportation progress in this century in terms of years of life span lost by accident death, distributed to the average individual in the population of the United States (table II). These values are approximately comparable to the preceding values based upon changes in physiologic age. (See p. 1108.)

In about the same way, we can tabulate the effects on life span of radiation received (table III).

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[12] This estimation of life span lost or gained is in terms of relative physiologic age change. Change in life expectancy may be estimated by determining life expectancy at a given age in terms of a given age + n years' change in apparent age. Thus, a person of age 40 has a normal life expectancy of 31.1 years. If his physiologic age is 50 (because of a sum of factors predicting —10 years age over the average), his life expectancy (from life tables) is 22.8 years, or an average loss of life span of 8.3 years. Thus the life expectancy lost in somewhat less than physiologic time lost.

TABLE III.—*Estimation of radiation effect upon health and life span*

Radiation received	Life shortening (in years)	
	If l. r. = −5 days [1]	If l. r. = −10 days [1]
(r.)		
50	−0.7	−1.4
100	−1.4	−2.7
200	−2.7	−5.5
400	−5.5	−11.0

[1] 2 columns are given because of uncertainty whether l. r. = −5 days or −10 days.

Thus it is observed that, although the estimated effect of radiation upon life span is a number worth attention, its magnitude of effect at low accumulated dosage is slight compared with many public health problems. It must be remembered that major problems such as smoking and overweight and fat metabolism are so subtle that they are estimated and established not by clinical methods but rather by statistical (actuarial) researches involving large population samples. The effect of smoking 1 pack of cigarettes per day, for example, appears equivalent in reduction of health and life span to the effect of between 200 and 400 r. of accumulated whole-body radiation. This is several times as great as the 50-r. limit currently recommended for occupational exposure; and 50 r., in turn, is on the order of 10 times as much as the individual would accumulate through fallout. If the life-span loss is estimated as 5 to 10 days per r. of whole-body exposure, the loss due to 50 r. falls within the range of −0.7 year to −1.4 years of life span. This effect is greatly exceeded by the magnitude of the smoking problem; the obesity problem; the problems of atherosclerosis, diabetes, and all the chronic diseases; the benefits of marital status; etc. The effect of 50 r. of whole-body exposure to the general populace can also be viewed as being in the same category of life-span loss as that which results in the population of the United States from use of the automobile. This estimation, however, does not include the problem of the mutation burden in the next generations following such radiation exposure.

SUMMARY OF THE FALLOUT PROBLEMS ON A GLOBAL BASIS

On a global basis, the fallout intensity of radioactive materials is no more than one-millionth of the high-level fallout that occurred by mishap in the vicinity of a thermonuclear explosion in the Marshall Islands in Ooctober 1954. Current estimations made directly in humans throughout 1953–56 place the fallout exposure from strontium 90 as being, on the average; sufficient to produce an irradiation effect of approximately 0.004 r./year to human bones. This is a small quantity of radiation—2 percent of naturally occurring bone radiation—and estimates of effects derived from this additional tissue burden will be correspondingly small compared with other human problems.

At the present time, according to the Libby report (October 1956), there is in the stratosphere about 2.2 megacuries of Sr-90,[13] and a similar quantity of cesium 137.[14] If all the material in the stratosphere (in the fall of 1956) were to descend upon the surface of the earth uniformly, the amount of either Sr-90 or Cs-137 would be about 12 millicuries per square mile. The time of retention by the stratosphere of highly dispersed fission products is on the order of many years. Measurements indicate approximately 10 percent fallout per year and 2.5 percent radioactive decay. As about 25 percent has been added to the stratospheric reservoir of dispersed fission products during the past 2 years, the level in the stratosphere has remained nearly constant over that time. The quantity of Sr-90 in the soil of the United States is somewhat greater than expected from the fallout estimated on an average global basis; in the far west it is

[13] Strontium 90 has a half life of 25 years and decays by emission of a β particle of 0.54 Mev. maximum energy to produce yttrium 90. Yttrium 90 is short lived (half life 65 hours) it decays to the stable zirconium by emission of a β particle having a maximum energy of 2.24 Mev. Because of the short half life of the daughter product and the probable insoluble chemical form of yttrium, the radioactivity of strontium 90 is equivalent to both its own beta decay and that of yttrium 90.

[14] Cesium 137 has a half life of 33 years and decays by β emission (0.52 Mev. maximum energy) with associated γ emission (0.66 Mev. energy.

23 m C of Sr-90 per square mile. This is due to the heavier fallout in the near vicinity of a nuclear exposion.

Strontium 90 distribution to September 1955

	m C/square mile
Worldwide except the United States and Pacific Islands	3. 4[6]
United States, except Utah, Colorado, New Mexico, and bordering regions	4. 9[6]
	12. 5[5]
Utah, Colorado, and New Mexico	20–23

The specific ratio of Sr-90 to normal calcium is a convenient way of expressing the Sr-90 problem.[15] This is because strontium closely follows calcium in chemical behavior. The levels of Sr-90 directly measured in young human bones during the period up to October 1956 are in the vicinity of 0.0038 r/year to the bone. Strontium 90 is deposited preferentially in the bone by a factor of more than 100 over the soft tissues, so that only the bones need be considered with regard to this isotope.

The Libby report estimates, on the basis of a balance between accumulated fallout of Sr-90 into the soil and uptake by cattle and man, that in America the human ratio of Sr-90 to calcium may eventually become 10 to 30 percent of that observed in the topsoil. The report estimates that Sr-90 now held by the stratosphere, in descending to the earth over the next 4 years, will produce a human Sr-90 concentration of from 0.016 to 0.038 r./yr. (0.004 to 0.010 MPC[16]), assuming that no further Sr-90 is added. The range of this expected gain of radiation exposure is equivalent to the extra cosmic radiation exposure experienced by individuals dwelling at altitudes of 5,000 feet (e. g., Denver, Colo.) compared with individuals at sea level. The estimation assumes that there is a selection factor[17] favoring calcium over strontium in uptake from soils into the plant and into the cow and into the human bones, so that 70 to 90 percent of the soil strontium is rejected in favor of calcium.

Both human adults and stillborn babies have similar concentrations of Sr-90 (i. e., similar Sr-90/Ca ratios). This is to be expected, since the developing child draws its calcium from the maternal calcium pool, which is in partial equilibrium with maternal bone. Both these human sources of measured Sr-90/Ca have been placed during 1954 and 1955 at approximately one-sixth of the value for cow's milk; the resultant adult human bone irradiation value for this period is about 0.0019 r. yr. (0.0005 MPC) from the Sr-90 content. Reported values for adults

[15] A convenient concept, established by relating irradiation of bone to bone cancer, is that a maximum permissible concentration (1 MPC) of strontium 90 is equal to 1 μC Sr-90 per 1,000 grams of calcium. The concentration of calcium in the bones is such that 1 MPC can also be expressed as 1 μC Sr-90 per 7,000 grams of bone. The concentrations of radioactive strontium are usually expressed in units of 0.001 MPC; the equivalence is 0.001 MPC = 1.4×10^{-7} μC Sr-90 g of bone, corresponding to 0.0038 r/year.

[16] MPC = maximum permissible concentration.

[17] Harrison et al. have evidence that elemental strontium-to-calcium ratios, compared in food, blood plasma, and bone, are strikingly different; for man they are:

	g Sr/g Ca	Proportional units
Food	17×10^{-4}	7
Plasma	4×10^{-4}	2
Bone	2.5×10^{-4}	1

This is confirmed by Comar in observations using radiostrontium and radiocalcium simultaneously added to the diet. In Comar's observations for milk, the discrimination achieved against strontium in the deposition ratio of Sr/Ca may be less than that for other food sources, in which strontium and calcium may have different chemical binding.

The problem of a protective discrimination for humans against the uptake of the maximum Sr-90/Ca ratio is presented in the Libby report. At this stage of understanding, this apparent reduction of Sr/Ca in bones of humans compared with soil, plants, or animals seems to reside partly in the large calcium pool of the adult cow's body, which constantly dilutes incoming strontium and calcium so that milk, at present, is always intermediate in Sr/Ca ratio between the cow's bones and the forage. Similarly, the human calcium pool dilutes incoming Sr/Ca (largely from milk products) so that human bones at this time always have a lower Sr-90/Ca ratio than cow's milk or cow or calf bones. The content of children's bones is much higher than in adult or stillbirth material. There is some evidence for atomic discrimination between strontium and calcium, but the problem needs further study to determine how much of Sr-90 uptake by bone is lessened at fallout equilibrium. If only dilution operates, with little or no discrimination, humans will develop a higher Sr-90 level than is now expected.

did not exceed 0.004 r. yr. in the sample studied, except for one individual measured at 0.008 r. yr. This is a very small number in terms of radiation effect.

If, in the fallout to be expected, the discrimination against Sr-90 in its course from soil to plant to human bone is by only a factor of 50 percent instead of a factor of 70 percent to 90 percent, Libby's estimate of the future Sr-90 concentration would have to be increased to 0.075 r. yr. (0.020 MPC), based on the present stratospheric and soil burdens. This level of Sr-90 would represent an additional radiation exposure to the bone, equivalent to the additional cosmic radiation experienced by those who dwell at 10,000 feet in this latitude.

Libby has estimated, from soil calcium levels, that if the entire Sr-90 burden reached the soil and humans came into equilibrium with the top 2 inches of average soil, humans would eventually approach a maximum value of 40 $\mu\mu$C Sr-90/g Ca, or about 0.15 r. yr. of bone irradiation. Such a value would approximately double bone irradiation over natural radiation.

ESTIMATION FROM HUMAN BONE ASSAYS OF FUTURE HUMAN BONE CONCENTRATIONS OF STRONTIUM 90

The uptake of Sr-90 has been directly measured in human bones as a function of age, and of location and time of collection (Libby, (5) Kulp et al.). The following summary conclusions can be drawn from analysis of this information:

1. Strontium 90 content of the bones in human stillbirths is increasing and, on the average, is estimated from Libby as follows:

United States of America	$\mu\mu$C Sr-90/g. Ca	Percent increase per year
December:		
1953	0.14	----------
1954	.30	114
1955	.66	120
1956	[1] (1.3)	(100)

[1] Extrapolated.

2. The bones of stillborn humans have a much lower Sr-90 content than those of year-old children. The Sr-90 content of children's bones, which may be averaged from the Libby report, is given in table IV. This table is representative of the Sr-90 concentration observed in children of early ages at two study intervals, namely September 15, 1954, and August 1, 1955, average collection dates. Newborns (stillbirths) have a much lower Sr-90 concentration, because the uterine source of Sr-90/Ca has some intermediate value between dietary Sr-90/Ca and adult tissue-bone Sr-90/Ca. The value for stillbirths, as of January 1955, is 0.31 $\mu\mu$C Sr-90/g Ca; at this same time, growing children, age 0 to 5 years, are laying down Sr-90 at 2 $\mu\mu$C Sr-90/g Ca. Thus, the fetal tissues appear to have available to them only 0.31/2=0.16 as much Sr-90 as the growing child. This is a reasonable fraction, considering the lesser relative amount of milk products consumed by the average mother and the fact that her tissue stores of calcium are largely from the prefallout era. The growing child at each interval of growth (i. e., 0 to 1 year, 1 to 2 years, etc.) dilutes the entering Sr-90/Ca by the existing quantity of Sr-90/Ca already present in the body. However, analysis of the increment increase in Sr-90 content shows that children of all ages are consuming and laying down equivalent concentrations of Sr-90/Ca, and that in January 1955, this concentration was approximately 2 $\mu\mu$C Sr-90/g Ca.

On this date, three sources of milk showed the following ratios:

Radiostrontium content of milk samples, January 1955

	$\mu\mu$C Sr-90/g Ca
Foreign cheese (5)	2.0
Chicago milk (5)	1.9
New York milk (7)	1.6

Since growing children have milk as their chief source of Sr-90, it is as expected that the value of milk closely approximates the concentration of Sr-90/Ca being deposited in growing bones. These values imply that, should milk remain as it was in January 1955, all children born close to this date

will eventually have in their bones an average concentration of Sr-90 of 2 $\mu\mu$C Sr-90/g Ca.

However, the milk Sr-90/Ca is increasing, and has been increasing since monitoring of milk was begun in 1953. Eisenbud's report [16] gives the following.

TABLE IV.—*Strontium 90 content of cildren's bones (from Libby report)*

Age	Weight		Sr-90 content		Sr-90 content in newly formed bone [1] (corresponding to January 1955)
	Average at measurement	Δ/yr.	Aug. 1, 1954, to Nov. 1, 1954	June 1, 1955, to Oct. 1, 1955	
	kg	*kg*	$\mu\mu$C *Sr-90/g Ca*	$\mu\mu$C *Sr-90/g Ca*	$\mu\mu$C *Sr-90/g Ca*
Birth	3.3		0.25	0.53	
1 year	7.2	3.9	.54	1.16	2.2
2 years	9.6	2.4	.43	.87	2.1
3 years	11.5	1.9	.39	.68	2.2
4 years	13.4	1.9	.35	.54	1.7
5 years	15.1	1.7	.33	.44	1.3
Average					1.9

[1] See the following:

$$\frac{\Delta\ \mu\mu C\ Sr^{90}}{\Delta\ g\ Ca} = \left[\frac{Sr^{90}/Ca_{(t_2)} \times wt_{(t_2)} - Sr^{90}/Ca_{(t_1)} \times wt_{(t_1)}}{\Delta\ wt_{(t_2-t_1)}}\right] \times \frac{12.0.}{10.5}$$

(The 12.0/10.5 is the correction factor for 10.5-month time interval Sept. 15, 1954, to Aug. 1, 1955.)

Sr-90/Ca content of milk in the New York area

Date:	$\mu\mu$C *Sr-90/g Ca*
June 1954	1.2
January 1955	1.6
June 1955	2.0
January 1956	2.7
September 1956	5.0

The minimum estimate of future average human burden of Sr-90, then, is that 5 $\mu\mu$C Sr-90/g Ca will be present in the bone. This corresponds to the latest reported value for milk concentration and to the fact that bone acquisition of Sr-90/Ca in growing children is very similar to milk Sr-90/Ca.

A difficult current problem is the estimation of future Sr-90/Ca in milk. The level of Sr-90/Ca in milk is increasing, and, by linear extrapolation, may be expected to raise the Sr-90 concentration in a year's time (by September 1957) to about 7 $\mu\mu$C Sr-90/g Ca. At this date, accumulated fallout of Sr-90, based upon the quantity estimated at the time of the Libby report, may be about 25 percent [6] to 50 percent [5] of the amount initially dispersed in the atmosphere. Since the Libby report was written, other nuclear detonations have occurred, so that it would be very reasonable to assume that fallout; by some 10 years from now, should have increased milk levels significantly. For lack of better information, we may assume a factor of, say, 3 to 5 times as much as September 1957 (allowing for residual hold-up in the atmosphere and for decay of Sr-90). Thus, milk levels and human bone levels by 1967 may be 20 to 35 $\mu\mu$C Sr-90/g Ca.

An additional factor must be considered, which may require that these future estimates be even higher. Cows, in body content of Sr-90/Ca, may be expected to lag several years behind the plant and soil levels. This is because of the large calcium reservoir in their bones and other tissues, and bcause the start of growth to milk-producing stage preceded current time by 4 or more years; moreover, the food consumed by dairy cows is customarily stored for many months before it is eaten. It is difficult to estimate that point in fallout time that corresponds to current milk values; it seems likely, however, that the Sr-90/Ca content of the bones of pasture-fed calves approximates the Sr-90/Ca level that adult cows

[16] Merril Eisenbud, Global Distribution orf Radioactivity From Nuclear Detonations With Special Reference to Strontium 90, Washington Academy of Sciences, fall symposium, November 15, 1956, Washington, D. C.

would secrete in their milk, were they in more rapid equilibrium with fallout. The following table, derived from values averaged from Libby's and Eisenbud's reports, shows that calf bones are approximately 60 percent higher in Sr-90/Ca content than milk. Thus, future estimations of Sr-90 levels should be at least 60 percent higher than the 20 to 35 $\mu\mu$C Sr-90/g Ca estimate, or, say, 30 to 50 $\mu\mu$C/g, in round numbers.

Strontium 90 content of various materials (in $\mu\mu$C/g Ca)

	Mid-1953	Mid-1955	Mid-1956
Milk	1.1	2.1	3.6
Calf bones	1.4	3.5	5.7
Alfalfa (Wisconsin)	6.7	18.0	----------
Soil (Wisconsin):			
2 to 6 inches depth	----------	9.0	----------
0 to 2 inches depth	----------	35.0	----------

It appears that the Sr-90/Ca of cow's milk is a close index of the concentration of strontium in newly acquired human bone. Current milk levels suggest that children's bones in the next decade will approach an average concentration of approximately 50 $\mu\mu$C Sr-90/g Ca. This is in close agreement with the estimation by Libby of a minimum average concentration of Sr-90 of 10 to 40 $\mu\mu$C Sr-90/g Ca. These estimates do not consider local variance in the United States, nor, with respect to future concentrations, the special problem of high-rainfall or low-calcium areas.

The upper value of approximately 40 $\mu\mu$C Sr-90/g Ca has been set by Libby upon the consideration that this is the projected specific concentration ratio when all the fallout is complete and mixed with the average calcium content of 2 inches to topsoil. There does not seem to be a way of independently confirming the upper average limit of radiostrontium concentration from observation of milk or bone. The biological concentrations are increasing rapidly with respect to time, approximately following the level of total accumulated fallout, and 40 $\mu\mu$C Sr-90/g Ca may not truly be a limit.

Whatever the speculation concerning future levels of Sr-90 in humans, we can be certain that current values (1956) represent a low level. If we translate a small dose such as 0.0038 r./year (0.001 MPC) into numbers predicting an increase in leukemia mortality (an estimate may be based upon tentative data that leukemia tendency may be doubled by 50 r. whole-body exposure [19]), an increment of

$$\frac{0.0038 \text{ r./yr.} \times 50 \text{ years mean life span}}{50 \text{ r./tumor doubling}} = 0.004,$$

or 0.4 percent increase in leukemia, is estimated. Since there are only approximately 8,000 cases of leukemia deaths reported in the population of the United States per year (plus 2,000 cases of bone-tumor deaths, which may be similarly affected by radiation), such a radiation burden is equivalent to an increase of 40 cases per year after 50 years' equilibration with this level of fallout. If radiation fallout and uptake of Sr-90 in human bones were to increase by a factor of 10, one could estimate 400 additional cases of bone tumor and leukemia induced per year after a 50-year period, in comparison with 1 million deaths from all causes and 10,000 expected deaths from leukemia and bone tumors. Both above numbers are small in comparison with overall public health problems.

Although there are some sizable uncertainties regarding Sr-90 burdens during the next 10 to 20 years, it seems from the average human values that Sr-90 may increase and become a public health problem if levels should rise to 50 $\mu\mu$ CSr-90/g Ca (equal to about 0.2 r./yr. to bone). There is time—but not much time—for a reevaluation of many unsatisfactorily estimated aspects of this problem, including the extent to which radiation exposure induces leukemia and bone tumors, and more precise estimation of the strontium levels in humans.

[19] This number may be high, since it is based upon whole-body radiation exposure, while induction of leukemia by Sr-90 exposure is the result of direct irradiation of bone and marrow, the specific tissues involved in the leukemia change.

At the reference level of 1 MPC of Sr-90 burden, which is 4 r./yr. to bone, an estimated increase in bone tumors and leukemia is

$$\frac{4 \text{ r./yr.} \times 50 \text{ years}}{50 \text{ r. per doubling of incidence of tumors}}$$

or an approximately fourfold increase in natural expectancy of these neoplasms with respect to the radiation-related component of their origin. This level may be reached by humans as a result of Sr-90 fallout. At some such value, reason argues against further exposure. The 1-MPC value based on radium exposure is consistent with a prediction of a fourfold increase in natural incidence of tumors. It would be difficult to observe a fourfold increase above natural incidence of bone tumors in animal-colony studies with radium, but not at all difficult in large human populations.

In summarizing their opinion for the British Report Cmd 9780, Mayneord and Mitchell write, "It appears however that each unit quantity of radiostrontium absorbed by bone confers a certain probability of bone-tumor formation, the tumor development time perhaps decreasing and the tumor incidence increasing with the dose. On the whole, the experiments seem in favor of a proportionality between the frequency of tumors produced in a given length of time and the amount of radioactive material in the body even at low-dose levels."

The problem in the experimental animal is that the frequency of bone tumor appearance is so slight that statistically significant increases in the frequency are not to be expected as a result of irradiation. The human problem is similar in that osteogenic sarcoma and leukemia are relatively unlikely occurrences, together causing about 1 percent of adult deaths in the United States, so that a small percentage change in incidence caused by radiation could not be distinguished from random fluctuation, and a relatively large fractional increase in the number of these cancers would not appreciably increase the total death rate.

No gross evidence of osteogenic sarcomas has been observed following administration of P-32 (approximately 100 rep to bones) to polycythemia vera patients. However, these patients do have a high incidence of leukemia. This leukemia tendency is probably attributable to both the radiation exposure and the nature of the basic disease of the blood-forming system in these patients.

Special phases of the Sr-90 problem need additional examination:

(*a*) In several areas of the world, Sr-90 concentration exceeds the average world values by more than a factor of 10 (4, 5). This excess poses questions as to the origin of the enhanced concentration. To a reasonable extent, it is explained by Libby as calcium deficiency of soils in such areas. Rainfall variation also leads to variation in fallout. It will be useful to know more about these anomalous effects. Current worldwide sampling is perhaps far from representative of the world as a whole, because special effort was made to seek out low-calcium high-rainfall areas.

(*b*) There may be a factor-of-8 difference between Sr-90/Ca concentrations in soil and in humans, resulting from discrimination in favor of calcium (Libby); this must be further studied.

(*c*) Some factor of uncertainty must be allowed for in the prediction of levels today and in the early future of Sr-90 in humans, considering that the most recent of these measures are based on early 1956. These uncertainties may amount to a factor of somewhat more than 10.

(*d*) Although it is unlikely that all these factors would reach their maximum, nevertheless, the total uncertainty in the estimated human burden of Sr-90 throughout the world could mean an upper limit of $10 \times 8 \times 10 \times$ Libby's lower estimate of exposure in the near future, 0.02 r./year, which works out to about 15 r./year or 4 MPC. This possibility indicates that the Sr-90 fallout problem urgently calls for further attention.

CESIUM-137 FALLOUT

The Cs-137 problem is quantitatively similar to that of Sr-90. These two fission products are present in the air and in fallout in approximately equivalent quantities (5), and they have similar decay rates. Whereas strontium is a bone seeker, cesium is found in approximately equal quantities throughout the body, though less in bone than in soft tissues. Its distribution roughly approximates that of potassium. Furthermore, cesium is not retained by the body. Thus, the cesium burden at any given time rapidly reaches equilibrium with the rate of fallout, in the potassium pool in plants and animals.

Marley, in the British report (4), writes (p. 124), "The highest body-activity detected so far in the United States is found to be $4\times10^{-3}\mu C$. This activity if maintained would produce a total body irradiation of 0.0006 r per year or about one-thirtieth of the dose due to naturally occurring potassium–40 in the body." Since this time in early 1956, the fallout level and fallout rate of Sr–90 have been increased only slightly, so that we may assume that the Cs–137 level in man, which is more reflective of immediate fallout, may have risen by as much as a factor of 2. It should remain nearly at this level, estimated as a maximum of 0.0012 r/year, for an indefinite period.

Cesium–137 body burden at 0.001 r/year is certainly not to be considered an adult hazard. With a linear relation between effect and dosage, 0.001 r/year over a lifetime would be less than 0.1 r, and irreversible accumulative effects of radiation, such as leukemia, might be increased by less than

$$\frac{0.1\text{ r.}}{50\text{ r. leukemia-doubling dose}}=0.002\text{, or 0.2 percent.}$$

Stated in terms of life span lost or of the total tendency toward disease, 0.1 r Cs–137 dose $\times$—10 days of life span per r amounts to 1 day lost from the life span. A loss of 1 day is very small compared with health-modifying factors that are measured in years instead of days. Thus, in comparison with the smoking problem, the long-term effect of Cs–137 is approximately one-forty thousandth as deleterious. Only this extraordinary method of estimation by extrapolation of effect can convince the human reason that there is any such effect at all; even the best statistical procedures could not detect it through study of the most accurate data on the 160 million people in the United States. A 0.2 percent increase in leukemia (which is approximately $0.002\times8{,}000$ cases per year) is just 16 additional cases. This 8,000 expected normal incidence can fluctuate by random interplay of chance factors by plus or minus 1 percent, equal to 80 cases per year; thus, 16 cases of increased incidence cannot be detected.

THE LEVEL OF RADIATION EXPOSURE FROM FALLOUT

The total increase in background radiation on a global basis, as a consequence of radioactive fallout, has been very slight. In the preatomic age, natural sources of radiation produced an average radiation exposure of 0.1 to 0.2 r/year. The variation is due to slight geographic differences, to differing radioactive content of earth and buildings, and to the variation of cosmic radiation with altitude. At 5,000 feet above sea level, cosmic-ray intensity (measured by numbers of ionizations produced in matter) is increased to 1.5 times the sea-level intensity of cosmic rays; at 10,000 feet, the cosmic-ray ionization is 8 times that at sea level.

The increased human-tissue irradiation due to fallout and ingestion of radioisotopes is approximately as follows:

	Soft tissue irradiation (r/year)	Bone irradiation (r/year)
1955–56:		
Cs-137	0.0009	<0.0004
Sr-90	0	[1] 0.002 [2] 0.004
Predicted future values:		
Cs-137	0.0012	<0.0006
Sr-90	0	0.04 to 1.5

[1] Adult.
[2] Young.

Table V lists human radiation exposures from a number of sources.

TABLE V.—*Human radiation exposure (r./year)*

A. EXTERNAL EXPOSURE TO WHOLE BODY

Natural radiation exposure		Exposure from fallout from nuclear detonations	
1. Cosmic radiation at—		1. Fallout to earth, October 1952 to September 1956:	
Sea level	0.038	Salt Lake City	0.050
2,000 feet	.043	All other United States cities and other countries	.003
5,000 feet	.056		
10,000 feet	.112		
15,000 feet	.214		
2. Radiation from the earth:			
England	0.04		
Berkeley hills	.12		
Sweden	.09–0.16		
Average	.10		

B. INTERNAL EXPOSURE

	To gonads and soft tissue	To bone		To gonads and soft tissue	To bone (1955)
Potassium 40	0.020	0.005	Strontium 90	(1)	[2] 0.002
Carbon 14	.001	.001			[3] .004
Hydrogen 3	(1)	(1)	Cesium 137	0.001	(1)
Radium	.002	.120	Iodine 131	(1)	(1)
Average	.023	.126	Average	.001	[2] .002 [3] .004

C. TOTAL EXPOSURE

Prefallout:			Now:		
At sea level	0.164	0.264	At sea level	0.165	0.269
At 5,000 feet (Denver)	.179	.282	At 5,000 feet (Denver)	.183	.287

D. INTERNAL EXPOSURE UNDER OTHER CONDITIONS

1955:			Predicted for individuals born now (if no additional nuclear detonations):		
Strontium 90:			Minimum average value predicted by Libby—0.004 MPC		0.016
Average value, USA		0.0019	Maximum average value predicted by Libby for United States—0.010 MPC		.038
Highest value reported		.0075	Certain low-calcium areas		0.16–0.38
Certain low-calcium areas			If humans at equilibrium should approach the Sr/Ca ratio of plants rather than 10 to 30 percent of plant Sr/Ca ratio		0.10–1.5
If humans at equilibrium should approach the Sr/Ca ratio of plants rather than 10 to 30 percent of plant Sr/Ca ratio			1956:		
Iodine 131:			Iodine 131	<0.001	
Thyroid	[4] 0.004		Probably	.0002	
Thyroid (maximum measured by Van Middlesworth)	[4] 0.004		Estimated	< .000001	
Other than thyroid (estimated)	<0.000001				

[1] Too small to be considered in this tabulation.
[2] Adult.
[3] Young.
[4] Possibly the true value is 0.001 or less.

The problems of radioactive fallout may also be examined in comparision with other ways of acquiring exposures to radiation (English values for radiological exposure are generally much less than in America (4)). (See table VI.)

Thus, it is possible that, from common use of X-ray-generating devices, the average person in the United States has already begun to accumulate an exposure to radiation effect that is sizable compared with the fallout problem. That no gross evidence of disease has become evident during these past few years of increasing radiation exposure does not disprove the existence of slight average effects of radiation. For example, at current estimation of leukemia induction by radiation, about 20 percent of the relatively rare cases of leukemia (0.5 percent of adult fatalities) may be attributable to natural radiation. There is no difficulty in believing that supplementary radiation resulting from our modern activities may have been responsible for the other 80 percent of known cases of leukemia; the average additional artificial radiation exposure per year would only have had to be 0.8 r. to account for this difference. Considering the generous use of unshielded and unfiltered X-ray equipment in dental offices and shoestores alone, and the lack of public and professional appreciation of need to minimize radiation exposure, it is even reasonable to conjecture that the addition of artificially created radiation exposure to natural irradiation may essentially account for leukemia. Faber has analyzed 828 cases of leukemia registered in Denmark in the period 1950–53 with regard to the amount and type of irradiation each patient received for 20 years prior to development of leukemia. The incidence of previous incidental X-ray or radiation exposure for the chronic lymphatic leukemia cases was 18 percent, for myeloid leukemia, 30 percent, and for acute leukemia, 32 percent. It appears that both acute leukemia and myeloid leukemia can be induced by radiation; and the traceable X-radiation induction may account for a sizable percentage of current cases in Denmark. Faber's information does not rule out that lymphatic leukemia may also be induced by radiation. The analysis of leukemia incidence in followup of three groups of individuals who had had varying exposures to X-rays or other radiation strongly suggests that the radiation induction of leukemia is proportional to the radiation exposure, and that for whole-body radiation exposure the number would be entirely consistent with an estimation that 50 r. doubles the chance of development of leukemia.[20]

TABLE VI.—*Common means of exposure to radium*

Source	Exposure	
	Directed to the specific body region	Scattered to the whole body (dose per use)
Routine chest X-ray [1]	0.05 to 2 r./exposure	
Fluoroscopic examination [2]	10 to 20 r./min	1/200 to 1/1000 of local dose.
Cinefluorography [2]	25 r. per examination	
Dental X-ray [1]	10 to 150 r. per whole-mouth series.	0.01 to 1 r.
Shoestore fluoroscopy [1 2] shoe-fitting unit	50 to 150 r./min. to feet	1 to 10 r./min.
Radium-dial watch [1] μ C/watch	7. r/yr. to the wrist	0.01 r./year.
Radium and X-ray [1 2 3] technicians (throughout the world).		p.1 to p.3 r./week, 5 to 15 r./yr.
AEC maximum permissible dose for 20 years' exposure.		15 r./yr. at 0.3 r./week.
Average accumulated exposure of 10 most highly exposed individuals over 5-year period—U. C. Radiation Laboratory.[4]		0.1 r./week, 5 r./yr.

[1] William Nolan.
[2] AEC Report to Congress.
[3] Jones.
[4] University of California Radiation Laboratory records.

RADIOIODINE FALLOUT

Of all the problems that we can currently evaluate, the radioiodine fallout problem is disposed of most readily. Radioiodine is produced in thousands of curies by some of the nuclear detonations, and, in falling to the earth's surface, it contaminates grass and is eaten by foraging animals. In its fallout, it is

[20] Court-Brown and Doll, Summary of Leukemia Induction, British Report (4), pp. 84–89.

greatly diluted and does not at any time become a human problem. The herbivorous animal, however, eats large quantities of grass; and in the cow, for example, essentially all the iodine 131 ingested accumulates in the thyroid gland. Over a few days' time, several hundred pounds of grass may be eaten, and all the iodine contained becomes concentrated in the 15 to 30 grams of thyroid tissue. Following nuclear detonations of the last 2 years, the thyroid concentrations of radioactive iodine in pastured cattle reached as high as 0.001 to 0.003 mμC/g (depending upon the quantity of fallout), and the average radiation exposure, as measured over 3 years, was about 1 r./year to the thyroid tissue. This would be of genuine concern to man at similar human burdens of I-131, because it is now known that thyroid tissue is especially sensitive to radiation induction of tumors. However, cattle fed principally in feed lots have only 1/100 (or less) as much I-131 as range-fed cattle. Further careful measurement of fresh human thyroid material has been routinely made during the last 2 years by techniques that are sensitive and reliable for estimation of I-131 content. Direct measurement shows that human thyroid, at any time of high uptake of I-131 by bovine thyroid, has less than 1/5000 of the bovine I-131 content. It is possible that human thyroids had less than 0.0006 mμC/g during the latter part of 1956, when range cattle had 1 to 2 mμC/g. It is certain that the human thyroid exposure during the 1956 period did not exceed 0.001 r./year, and the probable value is 0.00016 r./year or even less. (Interestingly, one human thyroid showed an activity comparable with bovine thyroid content of I-131; the case, when traced to its source, proved to be from a man who had previously been given a small tracer dose of I-131 in the Donner Laboratory. The observed quantity of I-131 was accounted for by the magnitude of the dose, the estimated excretion, and the radioactive decay.)

Up to this time, radioiodine from worldwide fallout is not a problem of concern to humans; and it is not expected that it will become a problem in the future.

SUMMARY

1. This paper reports a broad examination of the levels of radiation exposure incurred from fallout. The discussion is limited to Sr-90, Cs-137, and I-131, the only radioactive isotopes reported to become associated with human environmen in detectable quantities.

2. The worldwide effect of radiation from fallout is now far less than that of naturally occurring radiation from cosmic rays and from radioactive elements normally contained in earth, buildings, and body tissue. The inescapable minimum of natural radiation exposure, for all people, is about 0.1 r./year. The average person at sea level in the United States is probably receiving about 0.16 r./year.

3. During 1954-55 the Sr-90 concentration in human bones (both in adults and in stillborn infants) produced an average exposure to the bones themselves of 0.002 r./year. (Only the bones—not the soft tissues—are exposed to measurable levels of Sr-90 irradiation.) At current fallout trends, the irradiation of human bone by Sr-90 will increase to 0.016 r./year, perhaps even to 0.038 r./year (Libby). The maximum value projected in this discussion is 0.2 r./year. (These are average predictions for the northern hemisphere and for the major population densities of the earth.)

4. Radioiodine (I-131) activity has been measured in humans during periods of likely fallout exposures. Radiation exposure from fallout I-131 is essentially nil for humans.

5. Any analysis of the fallout of radioactive materials on a worldwide basis shows that it does not even remotely approach the threshold for acute radiation effects, which cannot be recognized below 100 r. in a single exposure. Radiation predicted from future fallout is still far less than natural radiation background. Increases in the internal radiation exposure of 0.1 r./year are not meaningful in comparison with acute radiation damage. Attempted comparisons are responsible for most misunderstanding of the fallout hazard to humans.

6. Life-span changes, cancer or leukemia induction, and cell changes appear to be proportional—as are genetic effects of radiation—to radiation exposure. Although these effects are not measurable in any individual exposed to fallout, they can be estimated, in terms of very small risks. The effects are dwarfed

in comparison with the adverse environmental hygienic factors that most persons regard as commonplace. For example:

Factor:	*Life-span loss per person (days)*
Smoking one pack cigarettes per day	3,000
Being 25 percent overweight	1,300
Having 25 percent elevated lipoproteins	2,500
Living in United States as a driver of an automobile	470
Working in industry (industrial hazard)	100

7. The evidence indicates that Sr-90 may eventually cause a worldwide increase in leukemia, accounting for about 2 percent of all deaths. Compared with the current accident rate, a 2 percent leukemia increase distributed throughout the entire population would be a life-span loss of about 1.0 year per person in the United States; all accidents account for a 2.3-year life-span loss per person, automobile use for 0.87 year. Thus the Sr-90 induction of leukemia is comparable with some of the mechanical mishaps we risk as a partial cost of the "advantages" of our mechanized and energized age.

8. The sum of evidence is that radiation has a deleterious effect upon man's health, but that the effects are extremely small at such slight radiation exposures as are involved in the worldwide fallout. Nevertheless, since radiation probably does affect man's health and progeny—even though minutely for minute exposures—incurring it should be treated as the equivalent of the spending of money or time, and should be allowed only for necessary gainful advantages.

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Interpretation of the Atomic Bomb Casualty Commission Report of Physical Growth of Hiroshima Children Exposed to the Atomic Bomb

Hardin B. Jones

Hiroshima children 5 to 19 years old (approximately 4,000, divided into 4 categories—males and females, exposed and unexposed) studied 5 years after exposure to the atom bomb are significantly shorter and lighter in weight than unexposed children, according to the report of Earl L. Reynolds to the Atomic Bomb Casualty Commission. Statistically the effect is slight in either sex, since weight is depressed about 2 percent and height is depressed about 1 percent (the standard error is 0.35 percent for groups of this size). Average individual variation in height or weight is about 12 percent (standard deviation) in Hiroshima children; therefore, the effect on height or weight of average radiation in the exposed group is approximately $\frac{\text{1 to 2 percent}}{\text{12 percent}} \times 100$, or about 10 percent of the effects of factors generally contribuating to lower-than-average individual differences in grows.

The effect of atom bomb exposure upon growth is measurably correlated with distance from the hypocenter of the explosion. The actual relationship observed is slight but significant, the correlation coefficient being about +0.1 for relationship of children's size and distance from the bomb. Since it is very likely that true exposure dose is rather poorly related to distance from the bomb at exposure and correction of this defective information should increase the correlation, the true effect of exposure upon children's growth is probably larger.

An improved estimation of quantitative relationship between exposure dose and growth in these children is possible by using groupings according to initial severity of symptoms, on the hypothesis that indeterminate shielding factors protected those who suffered fewer symptoms than would be expected from the theoretical dose. In comparisons by age and sex groupings, the asymptomatic groups were significantly larger in average physique, whereas the groups with definite irradiation exposure symptoms were significantly smaller. Such comparisons lead to a rough direct estimation of correlation between exposure and suppression of growth. Chi-square values show that the growth depression associated with exposure is significant. When chi-square values are converted to correlation coefficients, the result is equivalent to a correlation of approximately −0.4, indicating that the true relationship between irradiation exposure and growth, in doses sufficient to produce acute effects, may be larger than the above estimate; the square of the correlation coefficient indicates that 16 percent of the observed difference in growth is probably due to the effects of irradiation. Roughly estimated, the growth-depressant effect in humans should be equal to:

$$\frac{\text{growth change}}{\text{roentgen exposure}} = \left\{\begin{matrix}\text{correlation coefficient} \\ \text{of growth vs. exposure}\end{matrix}\right\} \times \frac{\text{standard deviation of growth}}{\text{standard deviation of radiation exposure}}$$

as derived from the regression equation. The computed value in this case is: $-0.4 \times \frac{\text{10 percent}}{\text{80 r.}} = -0.05$ percent per roentgen, all values being rough approximations.

The association observed between size and radiation exposure in Hiroshima is strikingly similar to that observed by Russell and Russell when mice were exposed to varying single doses of X-rays at 11.5 days after conception. Size of mice at birth decreased proportionally to radiation exposure from 100 r. to 400 r. The estimated effect upon embryonic growth per roentgen is 0.25 percent depression of size per r. It is noted that those children in Reynolds' study who were exposed in utero have twice the effect of decreased size seen in children exposed after birth; however, since the Reynolds study considered size 5 years after exposure rather than size differences at birth, as in the Russell study, the relative size difference at birth may have been even greater. Although the number of children irradiated before birth was too small to make the difference statistically significant, it is interesting to note that only a small correction factor would make the Hiroshima overall growth-depression ratio of 0.05 percent per roentgen comparable to the embryonic growth-depression ratio of 0.25 percent per roentgen observed in mice. These studies suggest that radiation effect upon both young mice and humans is the same with regard to suppression of growth.

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Representative HOLIFIELD. Now I suggest that we have a discussion.

We have certainly had a heavy diet today of philosophy on genetics and facts presented, and theories. I am sure now we can probably focus on some of the points we think should be focused on at this time.

I am going to ask Mr. Hollister to lead off with some questions.

DISCUSSION BY DRS. JAMES F. CROW, BENTLEY GLASS, A. H. STURTEVANT, HERMANN J. MULLER, W. L. RUSSELL, AUSTIN BRUES, AND HARDIN JONES

Mr. HOLLISTER. The first question, to Dr. Glass. Do we know specifically of any mutations in man that are radiation caused?

Dr. GLASS. No, you can't identify the individual mutations as being radiation caused or as having occurred spontaneously. The same kinds of mutations can occur through the action of radiation as may occur spontaneously. So you can't identify the particular mutations. There is aways a probability that even if the exposed person—the parent of the mutant person—received radiation, still the mutation might have occurred anyway. All we can go by is the increase in the frequency of mutations, which is thought to occur with a particular increase in dose.

Mr. HOLLISTER. So the radiation mutations produced in man or in any other organism are indistinguishable by kind.

Dr. GLASS. Exactly.

Mr. HOLLISTER. From any other sort of mutations.

Dr. GLASS. If we looked at the chromosomes we might find that a larger proportion of the radiation induced ones were visible losses, as has been brought out today. But just judging from the ordinary clinical symptoms, no, you could not distinguish them in kind from the spontaneous ones.

Representative HOLIFIELD. Are there any comments, or are you in general agreement? I see by your nodding that you are in general agreement.

Mr. HOLLISTER. I would like to ask, Dr. Muller, if you know of any mechanism seriously proposed that would predict a nonlinear effect for the mutation dose relationship.

Dr. MULLER. At high doses; yes. For what we call chromosome aberrations, but not for what we call gene mutations or point mutations.

Dr. Russell referred to a result of his and we, at our laboratory, obtained a similar one in flies, in which there was a less than linear apparent effect at very high doses, owing, as we judged, to the fact that the cells that had been worse hit were killed off more so that we lost the cases. But I do not see any way of getting a fundamentally nonlinear effect, especially at low doses. If the process takes place in any way like what we think it does, that is.

Representative HOLIFIELD. If any member of the panel wishes to comment, if you will raise your hand, I will recognize you. Dr. Russell.

Dr. RUSSELL. I should like to add to that that I think the possibility does still exist, if there is killing of cells at low doses. That is, down to a level at which cells are killed I think the possibility of departure from linearity might still exist. This level may be lower than we might tend to think from experiments on sperm. The spermatogonia seem to be sensitive.

Dr. MULLER. Might I add one word to that? If that is true, then it would work the other way from the way the people argue who believe in a threshold, because it would mean a relatively greater effect at low doses.

Dr. RUSSELL. Yes. I tried to bring this out, that it would be an increase in the effect, rather than less. But I do not think departure from linearity would be expected at such low levels as 0.1 roentgens, for I don't think there would be enough killing of cells at this dose level to make any difference. This would perhaps apply at the 10-roentgen level or something of this order. Down at the very low doses I would agree 100 percent with Dr. Muller that linearity would be expected.

Representative HOLIFIELD. Dr. Jones.

Dr. JONES. It is certainly true that several ionization events may be necessary for critical change within the cells. The ravages that can take place in the cell with time are so similar to radiation-induced events that even though one ionization occurs and several are needed, perhaps the preceding natural aging events have made the internal climate such that one ionization could produce that change.

In this sense we perhaps have a further reason for accepting the hypothesis of a proportional change down to very, very small doses. Of course, at these small doses we have very small effects.

Dr. RUSSELL. Could I add one more point to this? My point is that at the very low doses you would still expect linearity, but the slope of the line at these low doses might be steeper than would be predicted from our present data at the 300 and 600 r. points.

The question is, Have we measured the full effect at the present time? We think we need to measure the mutation rate at lower doses to be sure.

Representative HOLIFIELD. Thank you.

Mr. HOLLISTER. I would like to ask if it is not true that you as geneticists—when you draw a line between a good and a bad muta-

tion—do this almost entirely on the basis of how the mutation affects reproduction. Who would like to answer that. Dr. Crow?

Dr. CROW. From the standpoint of some theoretical computations that are made in assessing damage in the long-time future, this kind of an assumption is made. But in general, although we realize that most harmful mutations have some harmful effect on survival or reproduction, I think most of us—probably all of us—would be agreed that this is not really the point which as humans we are interested in. We are interested in the pain, disease, misery, all of which are associated with the reduction in survival value.

Mr. HOLLISTER. In other words, it is possible you could get into a situation, for example, where by process of continued mutation and selection, you could achieve a population that would reproduce very prolifically, but would have all sorts of other defects, that is, susceptibility to diseases and shortened life span and so on.

If we apply this criteria of good and bad mutation, you could do this?

Dr. CROW. As a very remote theoretical possibility. I don't think that is a likely possibility at all.

Mr. HOLLISTER. Dr. Crow, another question for you. This is the question of numbers of individuals limiting in the quantitative studies of natural mutative rates either in man or other organisms. Is the size of the population that you have to work with a limiting consideration in the kinds of experiment that you can perform or the kinds of data you can obtain?

Dr. CROW. In the study of human populations?

Mr. HOLLISTER. Either in humans, fruitflies, or mice.

Dr. CROW. I would say the numbers are the principal limiting factor. If one studies humans one cannot make experimental matings and that is serious. The organisms that are preferred for research purposes are those that you can grow cheaply and in large numbers, and that is why the fruitfly has been used.

Is that the point of your question? I am not quite sure.

Mr. HOLLISTER. My understanding is that you had postulated somewhere that there was some size or some number of individuals, perhaps a million or perhaps 10 million, that was a practical maximum size that an experimenter could work with.

Dr. CROW. I think you are confusing me with the U. N. committee. I cannot think of the man's name. Yes, Dr. Appleyard.

Mr. HOLLISTER. That is possible.

Dr. CROW. I think these are statements from Appleyard who has done some computations on the size of population that would have to be studied in order to detect differences of a certain small magnitude.

Mr. HOLLISTER. Is it not true that in some of these studies he has concluded that the populations have to be enormous?

Dr. CROW. That is correct. One can reach those conclusions even before seeing his figures.

Dr. MULLER. Could I interpose something since we mentioned the U. N. committee? I received information from someone in the audience in regard to a question that was raised or that I raised during my talk about the composition of the U. N. Scientific Committee. I stated that Dubinin had been scheduled to appear from Russia. I got information that he did not show up. Also, that he had not been

scheduled to be a delegate, but an adviser or consultant. Also, that at the most recent meeting there were various advisers present from other countries who were geneticists, not delegates, however. The geneticists who were delegates were confined to those I listed. Even though most of the discussion was done by the consultants rather than the delegates, it is an unfortunate situation if geneticists are not actual delegates, because although they were evidently given free rein during the discussions on the effects on posterity, nevertheless, as I indicated before, the effects on the exposed generation itself are also very important, and are very possibly closely related to the genetic subjects. Therefore, the committee should have geneticists to discuss those effects also.

Representative HOLIFIELD. Without objection, the record will be corrected.

Dr. MULLER. Yes, sir; it should be corrected. Our country had two good geneticists as consultants.

Mr. HOLLISTER. Dr. Muller, do you suppose that something analogous to the uncertainty principle in physics could exist in genetics with regard to this threshold question? For example, presumably we have to have an effect, tested in a certain number of cells. Leukemia, bone tumor, these have to involve more than one cell for us to observe. If this is true, does not this of itself indicate a possible threshold when in fact there might not be one. That is, we cannot observe a leukemia in 1 cell, can we, or a bone tumor involving 1 cell? Presumably it involves many cells.

Dr. MULLER. If the individual is unlucky enough by reason of the cell being lucky enough, then the one cell that became leukemic could give the individual leukemia.

Mr. HOLLISTER. But you would not know this until it involved more than one cell?

Dr. MULLER. No. There could be a mutation in one cell if our conception is right.

Mr. HOLLISTER. You would not know this experimentally?

Dr. MULLER. You would know it experimentally if that cell was in position in which it could express the tendency given to it by the mutation to divide or to reproduce or multiply in an uncontrolled manner. After a while it would crowd out the other cells and the person's blood would be full of these white cells, and he would have leukemia.

Mr. HOLLISTER. The thing that you would measure would be the presence of leukemia in the person after some of the multiplication had occurred.

Dr. MULLER. That is right.

Mr. HOLLISTER. So you would not know he had leukemia until it showed up as a result of many cells being pathologic?

Dr. MULLER. I think the question is on the same basis as all other genetic questions. All you get is a more or less random sample. You have to judge by that sample. That is why you need large statistics.

Representative HOLIFIELD. Dr. Glass has something to say on that.

Dr. GLASS. May I speak to that question, too? The more we press back into the knowledge of how the genes produce their effects, the more possible it becomes to detect the nature of those effects in individual cells. Thus even in a tissue culture of human skin epithelial

cells, if a mutation occurred in the one gene that we know of that controls the production of pigment, and changed it to an albino type, you would not have to have a thousand cells or a whole individual to know that mutation had occurred. You could spot it in that one cell.

If we knew enough about the biochemistry of different cells and enzymes, we could easily detect this in single cells.

Senator ANDERSON. Dr. Crow, do you agree with that? It looked as if you did not.

Dr. CROW. Let me get into this act, too. Back to what I think maybe you have in mind. At the present time we can demonstrate a linear effect perhaps down to 25 r. We could do a very large experiment and perhaps demonstrate a linear effect down to 10 r. We could do an enormously large experiment and demonstrate it down to 5 r. One cannot continue indefinitely. If that is what you mean by an uncertainty principle, I think there is something here. One cannot do a large enough experiment to demonstrate linearity down to an arbitrarily low value. Having said that, I would like to say that about this time we start relying on purely physical considerations of the kind that other people such as Dr. Pollard have been mentioning.

Mr. HOLLISTER. How about some of the testimony that Dr. Brues gave, which I am not sure I understood perfectly, and do not have in front of me—and if he is here, he might want to comment himself—to the effect that to cause cancer in body cells more than one cell would have to be affected by a dose of radiation before this effect would occur.

This might imply a threshold, although in fact a threshold might not exist.

Representative HOLIFIELD. Dr. Brues, have you been correctly quoted? Will you come forward? Pull up a chair and defend yourself.

Dr. BRUES. Was the question addressed to me?

Mr. HOLLISTER. I think you can help us first to make sure I paraphrased what I thought you said correctly.

Dr. BRUES. I shall in that case rephrase it to say this: I should not necessarily assume that a somatic mutation would be the basis of cancer a priori. But if it is, it still might be a little more complicated than the genetic situation, where just one cell is involved. I can think of at least two different ways in which that might occur, but I shall not take the time to go into them.

Mr. HOLLISTER. But these complications that you speak of involve the notion that more than one cell would have to be affected by the radiation; is that correct?

Dr. BRUES. That would be correct, yes. This is not proven or disproven, but it is a suggestion which I think is as likely as the other one which has been made, rather categorically.

Mr. RAMEY. Do the complications make for the threshold, then?

Dr. BRUES. I beg pardon?

Mr. RAMEY. Do the complications arising mean that you have to have more dose before you would get some sort of threshold effect?

Dr. BRUES. Yes. I think the suggestion I made rather specifically, and this was based on many things which have been observed in cancer pathology, rightly or wrongly, is the fact that cancer rather tends to arise in a tissue which has been generally disarranged. As for a general disarrangement or disturbance of the blood vessel supply of the tissue, this, I think, is not linear with radiation dose, but

like the erythema produced by irradiation on the skin, the old method of measuring dose, this appears to have some sort of threshold. If that is necessary as well as something else that radiation does, then we will not have a linear response. That is the point I made.

Representative HOLIFIELD. Is there any comment on that? Observing no hands raised, we will go to the next question.

Mr. HOLLISTER. Dr. Crow, do you know if experimentally, a population has ever been destroyed genetically?

Dr. CROW. I cannot think of an example. You mean by accumulating so large a number of mutations that ultimately it was killed off?

Mr. HOLLISTER. Yes.

Dr. CROW. Dr. Russell has reminded me of an example. One of Dr. Bruce Wallace's populations died out presumably as a consequence of very heavy radiation. Is the theoretical point here whether it is possible to induce a large enough number of mutations to kill off posterity without killing off the first generation?

Mr. HOLLISTER. Presumably this experiment proves it is possible.

Representative HOLIFIELD. I think we have time for one more question. We will allow each one of you, regardless of the time element, to comment on this. Are conclusions in the field of genetics being arrived at too far in advance of the data?

Dr. STURTEVANT. It seems to me, sir, that we have to draw some conclusions. We have to do something, because not doing something is equivalent to doing nothing. We therefore have to proceed on the basis of the best information we have. This is a common enough human experience; it happens to all of us every day. We have to reach decisions as to what to do or not to do without all the information we should like to have. I don't think that the situation here is any different from that which is usual. We have to proceed on the basis of the best information we can lay hands on.

Representative COLE. You concur with Dr. Crow when he said it is better to guess wrong than not to guess at all?

Dr. STURTEVANT. I think it is not only better to; it is necessary to. You have to make some kind of guess in order to live at all in this world.

Representative HOLIFIELD. Would there be any other comment on that?

Dr. JONES. I think the same thing applies to the question of life span effects. Here we simply do not know how life span effects operate in the small-dose range. We may be a long time in finding out information that applies directly in the small-dose range. In the absence of that, we have to make some estimate of the effect. If we relate this problem to fallout, we may estimate fallout to be now about 2 percent of natural radiation. Natural radiation is estimated to cause a life span loss of 25 to 50 days, if we live as long as 70 years. So the effect of fallout is only about 1 day or half a day on this basis, if we assume proportionality. This loss is extremely small but may be worth keeping in mind, even though it may be as small as one twenty-five thousandth of man's life span.

Dr. RUSSELL. I should like to make two comments on this. I think the geneticists on the National Academy committee have faced up to this problem, and in some sense should be complimented on this. I am a member of the committee, so I do not want to compliment myself, but the others. Most of them were experimental scientists and

they were very reluctant to come out with figures based on what they would consider, in some respects, inadequate evidence. However, it was necessary to face up to drawing a conclusion.

I don't think anyone should be reprimanded for drawing a conclusion when a conclusion was requested.

Representative HOLIFIELD. I certainly do not want you to think that this committee is reprimanding.

Dr. RUSSELL. No. Other people have.

Representative HOLIFIELD. It is very salutary that you brought this out. We believe it is in line with your scientific integrity to point out danger signs, whether you are sure how great they are or how many.

Dr. RUSSELL. I did not mean to apply my remark to this committee. I believe geneticists have been blamed for making too definite statements based on the evidence, perhaps mostly by medical specialists.

The other point I have is that we perhaps know more about this genetic hazard than we did about many other hazards we have experienced in the past. Many hazards man has been encountering were not known to be dangerous until many humans died from them. For example, many industrial poisons and even radium in the first place. I think the genetic hazard represents a situation where we know in advance a good deal more about it than we have done for some other things, including, I might say, some medical treatments. Some of these have been found to be hazardous only after several people have suffered from them.

Representative HOLIFIELD. Of course, the magnitude of the threat of nuclear radiation from war is the compelling factor in this matter.

Dr. RUSSELL. Yes.

Representative HOLIFIELD. Dr. Crow, I am sure you want to say something.

Dr. CROW. I agree heartily with the two people who have spoken.

Dr. GLASS. I agree, but I would like to add just one very brief comment. We know there is a genetics hazard. We don't know the exact amount of that hazard. We think that it is better to overestimate it than to underestimate it, and play safe, than to underestimate it and reap irreparable damage.

Representative HOLIFIELD. Mr. Ramey has a question, I understand.

Mr. RAMEY. I believe most of you gentlemen sat in on yesterday's discussion of the pathological or somatic effects. As biologists rather than geneticists, do you think that this linear effect that was brought out today for genetics applies in the somatic pathological field?

Representative HOLIFIELD. Who would be so bold as to answer that?

Dr. CROW. I will be so bold as to make an answer, but it will not be very definite. I believe most geneticists are convinced that at least some of the somatic effects of radiation are of a linear nonthreshold sort. I don't think anybody would be so dogmatic as to state that all such effects are or even what the fraction is.

Representative HOLIFIELD. Dr. Muller, what do you think about that? Would you have any opinion on that?

Dr. MULLER. My opinion is, as I said before, that the most important effects, those from which the human race when exposed to radiation suffers by far the most damage, and that is the shortening of

life effect, and probably leukemia and some related things, are in all probability linear without a threshold.

Might I also say with regard to the other question of whether we are going too far beyond the evidence, that it was not, of course, possible in these discussions to present the details of the evidence, and the reasoning involved, but that the estimates that were presented as what we regarded as probable were not in any sense guesses or speculations, but arrived at as a result of an enormous amount of work and calculations. Not only that, but that they were arrived at by more than one method. There was a totally different method used recently by a number of geneticists in arriving at the frequency of mutations in man. It was remarkable that at the end it was in very good agreement with the estimate reached by the first method that we had used.

I would make this qualification only of what Dr. Glass said, that we were not trying in the main to show the maximum effect. I would regard the preferred estimates as minimum estimates.

Senator ANDERSON. My question was not too serious an inquiry. I was just wondering if geneticists had a union, guild or gang, or something that teaches you to hang together? This is not only the most agreeable group of seven scientists, but certainly the most agreed group I have seen. I commend you of the fact that you have been able to hang together as long as you have through a rather long day.

Dr. STURTEVANT. I would like to say that I think it would have been very difficult to get together a group that would have disagreed with most of what has been said here among practicing geneticists.

Dr. MULLER. Might I take the occasion to thank the members of the committee on behalf of all of us for their having put on these hearings on this subject.

Representative COLE. Mr. Chairman, I am not sure that the question can be answered, but at least I am curious enough about it to pose the question.

Are there any other firm conclusions that can be reached based on data and experience with respect to the danger or hazard of radiation other than the one that was just voiced by Dr. Glass, that it does constitute a hazard? I am directing the question at all of them and I prefaced it by saying I was not sure it could be answered, but I was going to ask it anyway.

Dr. CROW. I find it hard to answer, Mr. Cole, because of the difficulty of deciding what we really mean by firm in a case like this. I think the conclusion that any effect of radiation is harmful is about as firm as a scientific conclusion ever is. Of course, the quantitative figures are much less firm.

Representative HOLIFIELD. From the field of physiology?

Dr. JONES. I would echo Dr. Crow's opinion that it looks as though we can definitely say that some effects do occur, and at very small dosages they are undoubtedly small effects. But they seem nevertheless to be effects, and we cannot say with certainty what the relative orders of magnitude of these effects are. I don't think there is any reason to be more concerned than to try to get better information as soon as possible. There is no public hazard at the moment compared to usual concepts of public hazard. We certainly do owe it to ourselves to find out what these effects are.

Representative HOLIFIELD. On behalf of the committee, gentlemen, I wish to express our collective thanks for this participation in this set of hearings. I am sure they will be read by a great many thousands of people with great interest. Your audience will be large. I think these hearings will be the year's best seller.

Tomorrow we will have Dr. William B. Looney as the leadoff witness, followed by Dr. Libby, Dr. Ralph Lapp, and Dr. Walter Selove, in the Senate caucus room at 10 o'clock.

(Thereupon at 5:10 p. m., Tuesday, June 4, 1957, a recess was taken until Wednesday, June 5, 1957, at 10 a. m., in the Senate caucus room.)

THE NATURE OF RADIOACTIVE FALLOUT AND ITS EFFECTS ON MAN

WEDNESDAY, JUNE 5, 1957

CONGRESS OF THE UNITED STATES,
SPECIAL SUBCOMMITTEE ON RADIATION OF THE
JOINT COMMITTEE ON ATOMIC ENERGY,
Washington, D. C.

The special subcommittee met, pursuant to recess, at 10:05 a. m., in the caucus room, Senate Office Building, Hon. Chet Holifield, chairman of the subcommittee, presiding.

Present: Representatives Holifield, Durham (chairman of the Joint Committee), Price, Cole, Van Zandt; Senators Anderson, Hickenlooper, and Bricker.

Also present: Professional staff members: James T. Ramey, executive director; George E. Brown, Jr., Hal Hollister, staff technical adviser, and Paul C. Tompkins, consultant.

Representative HOLIFIELD. The committee will be in order.

The subcommittee will continue its hearings on the effects of radiation on man, and today we will take a step back toward the pathology section of our witnesses. Our witness this morning is Dr. William B. Looney from the John Collins Warren Laboratories of the Huntington Memorial Hospital of Harvard University, Massachusetts General Hospital, who has been for the past 7 years conducting experiments and partaking in clinical work directly with patients who have had radiation damage. We should have had Dr. Looney on 2 days ago when we had the pathology section, but we could not fit him into the program because of the time limitation. We are mighty happy to have him with us this morning.

I notice you have quite an extensive statement here, and it is your purpose to submit this for the record, I understand, and to discuss the highlights of your statement.

Dr. LOONEY. That is correct, sir.

Representative HOLIFIELD. You may proceed, Doctor.

STATEMENT OF DR. WILLIAM B. LOONEY, MASSACHUSETTS GENERAL HOSPITAL[1]

Dr. LOONEY. Mr. Chairman and members of the committee, first, I should like to express my appreciation for the opportunity to

[1] Date and place of birth: March 18, 1922, South Clinchfield, Va. Education: Bachelor of science, Emory and Henry College, 1944; United States Naval Academy, 1941–44; doctor of medicine, Medical College of Virginia, 1948; internship, Presbyterian Hospital, Chicago, 1948–49; assistant residency, internal medicine, 1949–50; AEC postdoctoral research fellow in medical scence, National Research Council, Argonne National Laboratory, 1950–52. Work history: Assistant in medicine, University of Chicago, 1950–52; Radioisotope Laboratory, United States Naval Hospital, Bethesda, 1952–55; Officer in Charge of Radioisotope Technicians School, National Naval Medical Center, Bethesda, 1953–55; subcommittee on radiobiology, National Research Council, 1953–55; Consultant to Egyptian AEC, Cairo, Egypt, 1954–; specialist, Public Health Service Research, fellow of National Cancer Institute, 1955–; clinical and research fellow, Massachusetts General Hospital, 1955–; visiting fellow in physics, MIT, 1955–; consultant in radiation protection to Surgeon General, Department of the Navy, 1957–. (Submitted by witness.)

present this information to the Joint Committee on Atomic Energy.

As was just previously mentioned, my studies primarily have been on the effect of radiation in man, and the importance of the information to be presented this morning on the effect of radiation in man by radium is related to the fact that both radium and strontium are deposited in the skeleton similar to calcium. We have a considerable amount of information about the effects of radium in man; however, we do not on strontium. Since both strontium and radium act similarly to calcium, having the knowledge of radium, we can make estimates of the effects of strontium on man by our knowledge and factual data of the effects of radium on man.

I should like to confine my testimony primarily to the available knowledge we have on the effects of radium in man, and then at the end of the statement, based on the estimates from man, make some estimates as to the radiation dose from strontium necessary to produce similar changes in man over a 70-year period.

Man is constantly confronted with toxic agents in his environment which, if present in sufficient quantities, may produce temporary or lasting changes. By the accumulation of clinical and experimental information about the quantity of these toxic agents which produce minimal changes, safety measures are established. The present maximum permissible concentrations for radio elements in use today are based primarily on the results of clinical and investigative studies made over the past half century.

The principal sources of material for the study of the effects of internally deposited radioactive materials in man have been individuals who were employed in the luminous-dial industry, and persons who received radium for medical purposes. Radium salts had been given orally and intravenously for hypertension, arthritis, anemia, and other medical disorders from about 1915 to 1930. The painting of watch dials with luminous materials containing radium, mesothorium, and radiothorium started in this country in about 1914.

Adequate safety measures were not taken until about 1925–27, following the deaths from bone tumors, anemia, and crippling bone lesions, or all three, which occurred in some of these workers.

The first report in regard to the maximum permissible concentration, which I shall refer to as MPC, for radium was made by the National Bureau of Standards in 1941.

Senator HICKENLOOPER. I hate to interrupt you, Dr. Looney, unless you are willing to be interrupted in your statement.

Dr. LOONEY. That is perfectly all right.

Senator HICKENLOOPER. I notice the statement about the safety measures taken in 1925–27, following the deaths from bone tumors, anemia, and crippling bone lesions, which occurred in some of these workers. Do you comment later on the fact of whether or not all of the workers who received substantially the same dosage had the same results?

Dr. LOONEY. Yes, sir; this will be the context of the report.

Representative VAN ZANDT. Dr. Looney, when radium was first used, was there the same concern regarding the safety factor as there is today for radiation?

Dr. LOONEY. Well, this is a very fascinating history. The initial use of radium medically was based on the finding that radon, which is the first daughter produced by radium, was found in the waters of

the health springs in Europe, and a few biological experiments were done to show that radon or radium had beneficial effects. It was first believed that radiation had primarily a beneficial effect. As a result of the biological experiments and the finding of radon in the health springs, numerous people were treated. Historically, we now have the spectrum shifting from one side to the other.

Representative VAN ZANDT. One more question.

Dr. LOONEY. Yes.

Representative VAN ZANDT. I would like to ask about X-rays. Are you familiar with the development of X-ray from the time it was first used and was concern shown for the safety factor?

Dr. LOONEY. I think Dr. Taylor, who is in the audience here, would be certainly much more competent to comment on the general history. I think he is far more familiar with the overall details.

I will say, though, that certainly throughout the history of the uses of radiation, there certainly has been concern about its deleterious effect.

Representative VAN ZANDT. Thank you.

Dr. LOONEY. To go back to the report, the first report in regard to the maximum permissible concentration for radium was made by the National Bureau of Standards in 1941. Seven individuals having from 0.02 and 0.5 micrograms of radium in their bodies from 7 to 25 years had no noted changes referable to the deposition of radioactive materials. However, death had occurred in patients having as little as 1.2 micrograms of radium. The maximum permissible level of body burden for the amount of radium was established at that time as one-tenth microgram of radium. This was after the initial rapid elimination of radium from the body.

If radium is taken in, almost all of it is eliminated, but the fraction remaining in the bone is eliminated rather slowly. So the maximum permissible concentration for radium was set in 1941 at one-tenth microgram.

There have been two large studies, one made at the Argonne National Laboratory in Chicago, with which I was associated, primarily as coordinator in the evaluation of the clinical aspects with biophysical effects of the investigation. The other was made at the John Collins Warren Laboratories of the Huntington Memorial Hospital of Harvard University, Massachusetts General Hospital, Boston, and the Massachusetts Institute of Technology. These studies are continuing, and I have in the acknowledgments at the end made note of the people involved in these studies.

I would like to emphasize this was a very large study in which I was primarily interested in the clinical aspects, and I shall incorporate in the report the acknowledgment of the large group of people participating in these two studies.

This report is primarily a summary of these two recent investigations of people at or near the present maximum permissible concentration of radium.

Of the 78 patients that have been examined, the mean age of the luminous dial workers was about 21 years, and the mean age of the patients was about 34 years when they received radium. The average time of retention for both groups has been about 25 years with a

range of about 10 to 35 years. The average of these people in 1951 was about 50 years.

To summarize, we have information on people beginning about 15 to 20 years of age, and the major part of these patients have had radium in their bodies for 25 years. Many of these people are in the older age group, although the majority of the people are 50 or 60 years of age. We would like to have for a more complete picture additional information of people in the older age groups, and also people in the younger age groups. We have in the middle of the life span more information, but at either end the information diminishes.

Representative HOLIFIELD. Is there any information that the younger age group, with the same dose, is affected more deleteriously than the older age group?

Dr. LOONEY. Comparison of the effects of skeletal radiation in the unselected luminous dial workers and patients who received radium medically was made. It is not possible to draw conclusions on the present information in these two groups of patients. However, based on the available clinical information one might consider that the luminous dial workers were less affected after beginning employment at ages 15 to 20 years, than the patients who received radium medically in the 1930's.

Representative HOLIFIELD. Is that on comparative doses?

Dr. LOONEY. On comparative doses, yes, sir. As I say, I think this data is inconclusive. This is just based on the information we have available at present in man.

Senator ANDERSON. Do luminous dial workers go to work at 15?

Dr. LOONEY. Fifteen to twenty years of age; yes. Some of them started at 14 and 15. This was in 1915.

Representative HOLIFIELD. And they absorbed this radium, as I understand, by putting the tip of the brush in their mouths to point up the brush in order to paint the numerals on the watches. Was that the way they received it mostly?

Dr. LOONEY. I think this, plus the fact there was radium in the atmosphere and they inhaled it. There were no known effects at that time. In 1924 a dentist discovered destruction of the mandible of some of these people. This established the fact that the ingestion and inhalation of radium produced deleterious effects in man. This very tragic experience is the foundation upon which some of the maximum permissible concentrations of the most important radioelements in use today are based.

There is one other point I think should be brought out in regard to these patients. Ten of these fifty radium patients, and sixteen of the luminous dial workers were selected because of symptoms. In other words, they were discovered because they were having trouble. It is apparent that, if we had a thousand people and we saw only the people who were having difficulty, we would have a biased or selected group of people from the population of radium patients, and luminous dial workers. About one-third of these people were found because they had symptoms. This would tend for one to overemphasize the effect of ardioactive materials based on the present information. This is something the committee should keep in mind.

Thirteen of these seventy-eight patients having between one-hundredth and four-tenths micrograms of radium had no changes attributable to the effects of radium, with two exceptions. One patient

having 0.15 microgram of radium had minor areas of decreased density demonstrated roentgenographically in his teeth. These are minor changes; however, this patient was discovered because of the changes. These findings are characteristic of the dental changes in people we have seen with more radium in their body. The other patient was an 83-year-old woman who had very severe arthritis, was incapacitated, and had minor changes in her skeleton. Old age, plus the incapacity, might have contributed to the changes.

Sixty-five patients had between 0.5 and 23 micrograms of radium. All of these patients had either skeletal changes or symptoms attributable to radium, or both, which we could reasonably attribute to radium, with 3 exceptions; these people had between five-tenths and 1 microgram of radium. We should keep in mind the one-tenth microgram as the present MPC for radium, then we can mentally refer to the changes.

These patients began to have difficulty with about 10 times the amount of radium greater than the maximum permissible concentration.

Now I would like to go into the mechanism of deposition of radium in the skeleton, the pathological changes seen, and then relate these pathological changes to the amount of radium in the body. Radium is deposited in small areas of high concentration, so that we have an irregularity of deposition. This is an important factor to take into consideration, because in the last analysis we are interested in what happens in certain areas. If radium is concentrated in the area of bone tumor formation, then we are not as interested in the average value of total bone, as we are interested in these areas of concentration.

Analyses of samples of bone from some of these people have been made. In samples taken from different bones and from several parts, the same bones, we have found that these concentrations vary as much as by a factor of 10, and maybe greater. When we talk of average values of one-tenth microgram, we must realize that these people have areas of concentration which may be much greater than that. This has to enter into the consideration of the MPC of radium as well as strontium.

If you will go back to figure 2 at the end of my statement, there is an autoradiograph which shows how the radium is deposited in one of the bones. It is the picture here [indicating], showing the actual radium deposition in the bone. You will see that the dark areas represent areas of radium. (See p. 1176.)

In considering the effects of radium, you must consider the effect in the small areas of concentration in relation to the pathological changes.

Now, the changes that have been shown to develop in these people are areas of abnormal bone formation, which occur usually at the ends of the bone. When an X-ray is taken they show up as areas of increased density. The effect of radium causes abnormal bone to be produced, and it is generally considered that tumors develop in and around these areas of abnormal bone formation.

The other characteristic change is areas of decreased density in the shaft, or the middle of the bones, and these show up in the X-rays as areas of decreased density. When we examine people who have had a sufficient amount of radium, we see these areas of either increased or decreased density which are scattered throughout the skeleton, and

these characteristic changes are one of the most reliable clinical methods we have at present for determining the early effects, or the first detectable effect, of radioactive materials in man.

Chairman DURHAM. What kind of a dose, Doctor, was that received by the bone structure in figure 2 of your statement?

Dr. LOONEY. This patient had 1.3 micrograms of radium, sir. This is about 13 times the present accepted MPC for radium.

In figure 4 you will note that this is a very detailed microscopic picture of radium being deposited in one of the fundamental units of the bone, known as the Haversian system. You will note the alpha tracks from the radium coming from those two Haversian systems, and this relates to the black area you saw on the autoradiograph of the whole bone. This is a microscopic picture showing the radium deposited in the small area. (See p. 1178.)

Representative HOLIFIELD. Do you have that same picture on the screen?

Dr. LOONEY. Yes, sir, I do. This is the picture here [indicating]. I might point out that here [indicating] is the area of the increased density that you see at the heads of the bone here and here [indicating]. The areas of decreased density are in here and here [indicating].

The areas that you will notice here [indicating] are the areas of destruction in the bone, and this is an X-ray of the bone showing the areas of increased density and the areas of decreased density, and these are changes characteristic of radium deposition. It is the best clinical method we have of determining the effects of radium.

Representative HOLIFIELD. Is it your judgment that the strontium 90 would have the same effect?

Dr. LOONEY. It is the best means we have of comparing the effects of strontium 90 to the effect of similar radio elements in man, and we have to go on the best available evidence we have.

Representative HOLIFIELD. Have you taken pictures of bones of mammals that have been subjected to strontium 90 exposure?

Dr. LOONEY. This has been done in some of the atomic energy laboratories. I have not been directly connected with this, and I am sure that some of the people in the audience could comment on that. Strontium and radium and calcium are deposited in the skeleton in similar manner; this is the basis of comparing the radium data with the present level for strontium.

This [indicating] is the picture of the alpha track, or the actual radiation from radium in one of the fundamental units of the bone, and this [indicating] is the underlying bone, showing an area of destruction, and another area showing no change.

Here [indicating] it shows the normal bone, and this is a typical osseous tissue adjacent to the normal bone, which results in these areas of increased density seen roentgenographically. In animals we see this atypical osseous tissue formed following plutonium, strontium, and radium administration, and in man similar changes are produced by radium. We consider that the mechanism is similar in the production of these changes.

Chairman DURHAM. What kind of rays are those, Doctor?

Dr. LOONEY. These are alpha rays. This is actually a picture taken by photographic emulsion over the bone itself. In other words, the track was coming up in the photographic emulsion, and it is like

taking a photograph, and this is the photograph of the alpha track.

Representative HOLIFIELD. No one has used "micrograms" before in the presentation. Will you relate that to a microcurie?

Dr. LOONEY. Yes. The microgram of radium and the microcurie are the same, because the microcurie was established from radium. So if I speak of micrograms or microcuries, as far as radium is concerned it would be the same.

Representative HOLIFIELD. That is what I wanted you to say for clarification of the record.

Dr. LOONEY. Yes.

Representative HOLIFIELD. Are the alpha or gamma rays which might be emitted of the same intensity and range in strontium 90 as in radium?

Dr. LOONEY. No, sir, they are not, because most of the radiation from radium is alpha radiation, and the radiation from strontium is from beta radiation.

Now you have heard the term "relative biological effectiveness" used. The RBE of radium has not been taken into consideration in the determination of the present MPC for strontium 90. Tho RBE [indicating] may be more than unity [indicating], so that this would permit the raising of the strontium level based on the radium data, if we were to determine that the effectiveness of irradiation from radium was greater than strontium in producing biological changes. In other words, it is generally considered that the alpha rays are more effective in producing biological changes per unit of energy dissipated to the material. These may be inherent safety factors that can be incorporated in present considerations of the MPC for strontium.

Representative HOLIFIELD. So if there would be a difference, it would be weighted toward the greater effectiveness of radium?

Dr. LOONEY. Yes, it would be weighted. Any change would be to the raising of the strontium MPC.

I would like briefly to comment about the hematological changes in these patients. The changes have been minor, and the only changes we have found have been minor changes in the size and shape of the red cells. In a few exceptions there have been anemias that have occurred in these people, but the hematological findings have been very minor changes compared to the skeletal changes.

I should like to go on to symptoms, and probably I should define what I mean by symptoms in these discussions. I shall refer to symptoms as the time when the patient becomes subjectively aware of these skeletal changes which we see. In other words, we first see skeletal changes in many of these people who have no symptoms at all. When skeletal changes progress to the point that the normal configuration of the bone is destroyed, symptoms usually occur the patient may have a fracture of the femur, or he may have destruction of the hip, and he limps, then these denote symptoms. The patient is aware these skeletal changes are present.

In going over these patients, there have been 11 patients of the 78 who have had destruction of the hip, the head of this [indicating] hip bone, so that they walked with a limb. All of these 11 patients have had seven-tenths micrograms or more of radium in their bodies.

There have been five patients with fractures of the femur. Some of these fractures have occurred from very minimal trauma. I remember one patient we examined had a fracture of the femur after

her husband came to a sudden stop at a stoplight, and the pressure of her foot on the floorboard of the car caused this fracture of the femur. So that the skeletal system does become more fragile, after a period of time, with these people in this range.

Representative HOLIFIELD. How did that particular patient receive that does of radium?

Dr. LOONEY. This patient was a luminous dial worker, sir, and she worked at Ottawa, Ill.

If you will turn to chart 1, you will note that this is the summary of the X-ray findings of 32 patients who had a complete skeletal survey. These patients were arranged in the order of increasing amounts of radium in the body. The frequency with which these characteristic skeletal changes, which I have shown to you, have occurred, were plotted as a function of the amount of radium in the body. You will note from the left hand vertical bar that there were 15 patients having between five-tenths and 1 microgram of radium in the body. You will notice that about 15 percent of the total bones that could be involved had these characteristic changes. (See p. 1174.)

There were 8 patients between 1.1 and 2 micrograms of radium, and you will notice that the frequency goes up to about 55 percent, and that between 2.1 and 14 micrograms of radium it goes up to about 60 percent.

If you will turn to table 1, you will notice that in the first group of patients, the average age was 49; the average age of the second group was 52; the average time of retention in the first group was 20 years; the average time of the second group was 22 years; the average age of the third group was 61 years; and the average time of retention was 22 years. (See p. 1174.)

We have a group of people who are about the same chronological age, who have radium retained for about 20 years, in which we can show a correlation between the frequency of these characteristic changes and the amount of radium in the body.

I think this is probably one of the most significant clinical observations that has been made—the correlation an objective clinical change with the amount of radium in the body within a specific dose range. I want to emphasize that extrapolation of the result of this clinical data either one way or the other would present many difficulties.

Chairman DURHAM. How did these patients receive this, Doctor?

Dr. LOONEY. With two exceptions, all of the people received this for medical purposes.

Chairman DURHAM. For treatment of other diseases?

Dr. LOONEY. Yes, sir; for the treatment of other diseases.

An important point to bring out here is the fact that people with 10 and 15 micrograms of radium did not have a proportionate increase in skeletal changes. In other words, we did not see people with greater amounts of radium in the body having greater changes. This would be consistent with the hypothesis that the changes that we see are the result of the dynamic interrelationship between the destructive processes and the reparative processes of the body. If we had radium with some other destructive skeletal disease, we might see changes at a much lower level than if the patient had no disease. Radium, plus condition A plus condition B might produce changes at 1 microgram. Radium, plus condition B might produce changes at 5 micrograms, and radium alone might produce changes at 10 micro-

grams. This is consistent with the hypothesis that the body is constantly repairing itself from destructive changes. When the body can no longer repair these changes, then permanent changes occur, in regard to radium, the skeletal destruction can be seen on roentgenographic examination.

Representative HOLIFIELD. As a term of common reference, would you say that any effect upon the bone is usually referred to as a tumor or cancer of the bone?

Dr. LOONEY. No, sir.

Representative HOLIFIELD. Or is there a differentiation between those two?

Dr. LOONEY. Yes, sir; there is quite a differentiation between those two.

Representative HOLIFIELD. Will you explain that for the record?

Dr. LOONEY. Yes, sir. These changes which we have seen—these changes here [indicating]—are minor changes, and are from abnormal bone formation. The areas here [indicating] are from small areas of destruction in the bone, and these can be seen in other conditions but usually not with the distribution and the characteristics seen in the radium patients. In other words, this is not diagnostic of radium, but it is very characteristic, and there are very few other medical conditions which will produce the same picture. I might say other nonmalignant medical conditions.

Chairman DURHAM. Doctor, did any of these patients in this group develop cancer from the normal treatment of other diseases from this radium?

Dr. LOONEY. I want to go into the number of tumors that have developed in these people, sir. I could not say whether these developed tumors from other conditions. All I could say is that a very large number of these people developed bone tumors, and out of proportion to other groups of people.

Of these 78 patients, 15 people have developed tumors.

If you will refer to figure 14, you will see the distribution of tumors that have occurred in these patients. You will notice that they usually occur at the end of the long bones, in the same areas in which you see these areas of abnormal bone formation.

I would like to read from that portion of the statement (p. 1170) entitled "Bone Tumors."

The 15 malignant tumors which developed in the 78 patients recently evaluated were found in individuals containing from 0.5 to 10 micrograms of radium in their bodies. The patient having 0.5 microgram of radium was a luminous-dial worker. It is reasonable to assume this patient ingested mesothorium and radiothorium. The patient with the lowest radium concentration, who had received radium medically, had 0.9 microgram of radium. The patient with the lowest concentration of radium, which was considered not to be contaminated with members of the thorium decay series, and who had developed a bone tumor, had 3.6 micrograms of retained radium.

It is important to emphasize that the luminous-dial workers used not only radium, but mesothorium and radiothorium. We have only measured radium, so we have not taken into consideration the radiation dose from either mesothorium or radiothorium.

The accumulated radiation dose with 3.6 micrograms of radium was estimated to be about 5,000 rads during the 25 years from radium administration until tumor formation.

Some of these people were selected because of symptoms. The number of bone tumors in the patients which were not discovered because of symptoms were located in a review of the records. We find that there is a frequency of about 2 percent in this unselected group as compared to a frequency of 14 percent of the entire group of 78 people. Although this is not conclusive, I do think it should be considered as it suggests this may be a biased group of people with whom we are dealing.

Because of the major interest in the possibility of bone tumor induction by radiation, I have taken two comprehensive articles in which the investigators have summarized the cases in which bone tumors have been produced following external radiation.

Vaughan in 1956 reported 39 cases of sarcoma arising in bone following external radiation have been recorded in the literature. The latent period between receiving radiation and the development of the tumor was 3 to 11 years. The radiation dose was not known in all of these cases; however, in most recorded cases it was estimated to be usually greater than 3,000 roentgens (1,500 to 7,000 roentgen range).

Cruz et al. in 1957 reported an additional series of 11 cases in which the bone tumors developed from 4 to 24 years after external radiation. The total radiation dose given ranged from about 1,000 roentgens to 5,000 roentgens, and was given over a period varying from 1 month to 9 years.

I should like to comment now on the possibility of bone-tumor production from strontuim 90, based on this information of tumor induction following external and internal radiation in man.

The assumption is often made that the incidence of the effect of strontium 90 is proportional to the magnitude of the dose. This assumption has been used to estimate the bone tumors which may be produced from the low concentrations of strontium 90 in the skeleton from fallout. There is no exact evidence either proving or disproving this assumption, but there is some clinical evidence which suggests that this assumption is overcautious.

The 50 bone tumors which have been known to have been produced in man from external radiation, and reported, were summarized in the previous section. The skeleton in the localized area of tumor induction received at least 1,000 roentgens, and usually more than 3,000 roentgens of radiation. If it is assumed that the radiation dose to bone is greater by a factor of 2 than the measured skin dose—in other words, the radiation going into the bone would absorb more radiation than the dose measured at the skin. So we are assuming it may be more than a factor of 2, maybe greater than this, but we are saying this as a reasonable estimate—then the minimum observed carcinogenic dose from external radiation would be about 2,000 rads, with the majority of the tumors being produced by more than 6,000 rads of external radiation.

Representative COLE. I would like to ask Dr. Looney, Mr. Chairman, to explain his statement that there is some clinical evidence which suggests that the assumption is overcautious, the assumption being that the incidence of the effect of strontium 90 is proportional to the dose. Now what do you mean when you say "there is some

clinical evidence which suggests that this assumption is overcautious." In what way?

Dr. LOONEY. I am saying that the minimal carcinogenic dose that we have reported for tumors to be produced in man is in the order of 2,000 rads. Based on the present clinical evidence we have today, we cannot prove or disprove that smaller doses would produce tumors, but I am just presenting to you the present evidence we have in man known to produce tumors. I am just saying that this assumption might be overcautious.

Representative COLE. What do you mean by "overcautious"? That is what I want.

Dr. LOONEY. May I come back to this at the end of these comments? Maybe this will clarify this statement.

What I have attempted to do is to present the order of magnitude of radiation we are dealing with. The following estimates on strontium 90 are based on the assumption that strontium 90 will be present in the body over a life span of 70 years. If we assume it is in equilibrium with bone or there is a constant intake of strontium 90, it would tend to approach a uniformity in bone.

Estimates which we can make from this information about the levels of strontium 90 necessary to produce bone tumors, skeletal roentgenographic changes, and total skeletal radiation from background radioactivity are as follows: Ten microcuries of strontium deposited in the skeleton for 70 years would give an estimated dose of about 2,000 rads. This is the minimum radiation dose recorded which has produced a bone tumor in man. This should give some idea of the magnitude of strontium levels which may produce bone tumors in man. You will notice that 6,000 rads is the estimated amount of radiation known to produce most tumors. The amount of strontium 90 which would deliver 6,000 rads to the skeleton over a life span of 70 years would be in the order of 30 microcuries.

The minimum radiation known to produce tumors in man is in the order of 2,000 rads, which would be 10 microcuries. The present MPC for industrial workers is 1 microcurie, and this would give 200 rads over a 70-year period. (The tenth of a microgram of radium would also give about 200 rads.) The total dose from natural radiation, based on the estimates of Dr. Robert Dudley of Massachusetts Institute of Technology, from all sources of radiation to the skeleton over a life span of 70 years, would be in the order of 10 rads. The sunshine unit, 1 times 10 to the minus 3 microcuries of strontium, would deliver about two-tenths of a rad to the skeleton in 70 years.

I will leave this up on the board so you can refer to it as the order of magnitude, and I will try to keep all of the units in rads so that this will give a basis for comparison.

I should like to go back to my statement in the section titled "Comments on * * * Bone Tumor Formations * * *." (See p. 1171.)

The patient with the smallest total body radium known to induce tumor formation, in which the possibility of contamination of the thorium series is unknown, died from a bone tumor in 1952. The estimated total body radium was 0.9 microgram. The time after administration is unknown, however, it is reasonable to assume that it was about 25 years. Based on the estimates above, the patient would have received a total accumulated does of about 1,800 rads during the 25-year period.

Histopathological changes have been demonstrated by roentgenographic examination of the skeletons of radium patients prior to development of the tumors. It is generally considered that the bone tumors developed in or around areas of atypical osseous tissue formation. Bone tumors have been shown to develop in or around the abnormal bone formation in animals given plutonium, strontium, and other radio elements.

If it could be shown that these histopathological changes are preliminary steps to bone tumor formation, then it could be assumed that as long as the body reparative processes prevented the abnormal bone formation, bone tumors would not develop. It should be emphasized that the associated histopathological changes seen prior to bone tumor formation in the radium patients may be coincidental findings. Proof of a correlation must await a better understanding of tumor induction by radiation.

Now there is one other bit of information in which I must emphasize little reliance can be placed. However, it is the best available evidence we have in man. This information pertains to the latent period of tumor development in relationship to the magnitude of the dose.

In reviewing the latent period for tumor induction in the luminous dial workers reported by Martland in 1931, it was found that the latent period for tumor formation was about 5 to 10 years in 6 patients who developed tumors. Only 3 of these patients had estimates of total body radium reported, and these estimated were 6, 15, and 50 micrograms.

The 8 luminous-dial workers who were examined in the recent Boston-Chicago investigations, and who died from bone tumors, lived for an average of 25 years after beginning employment. The average total body radium was 3.4 micrograms—range of 0.5 to 10 micrograms.

We have very meager data, but there does seem to be an inverse relationship between the latent period and the radiation dose necessary to produce bone tumors. The estimates based on this would be in the same order of magnitude as the estimates we have by the known radiation dose that produces bone tumors in man.

Therefore, the available clinical information we have at present indicates that the radiation dose for bone tumor production in man from both internal and external radiation is in the order of 2,000 rads.

The present MPC for radium, as I mentioned previously, was established in 1941. The fact that we are finding changes now at four-tenths of a microgram, which is four times greater than the MPC, is probably offset by the confidence in the larger group of patients which we have studied for longer periods of time. It is reassuring that it has not been necessary to change the MPC of radium over this long period of time, in view of the large amount of information that has been accumulated since it was established.

I have listed some of the factors which may permit the raising of the present levels for strontium 90. As I have pointed out, estimation of the mesothorium and radiothorium content in luminous-dial workers is being carried out. If it is found that the mesothorium and radiothorium contribute significantly to the dose, this would permit raising the present levels.

The characteristic changes we have seen may occur in the normal population, and further studies may permit us to obtain a better

understanding of how these changes are produced, thereby permitting a raising of the MPC.

As I mentioned in the introduction, most of these people were studied beginning about 15 or 20 years of age, so we have a gap in our knowledge of the younger age groups, which might necessitate lowering the present level. The present MPC is based on our present methods, and knowledge of the clinical changes produced by radioactive materials. It is possible that other subclinical effects may occur which may necessitate lowering the MPC.

In summary, it is considered that the best estimates which can be made in regard to the effects of strontium 90 over a life span of 70 years on the present incomplete information on the effects of radium in man are as follows:

The skeletal content of strontium 90 necessary to produce a bone tumor in a life span of 70 years would be in the order of 10 microcuries of strontium.

The skeletal content of strontium 90 necessary to produce significant changes, such as destruction of the hip would also be in the order of 10 microcuries of strontium over a life span.

The skeletal content of strontium necessary to produce minimal skeletal changes, which were demonstrated roentgenographically, would be in the order of 2 microcuries of strontium.

It should be emphasized again that these estimates of the concentrations of strontium 90 which may produce skeletal damage are the result of estimates based on the available information at present.

Representative HOLIFIELD. Have you finished, Dr. Looney?

Dr. LOONEY. Yes.

Representative HOLIFIELD. Are there any questions of Dr. Looney?

Representative VAN ZANDT. Mr. Chairman?

Representative HOLIFIELD. Mr. Van Zandt.

Representative VAN ZANDT. Dr. Looney, you have spelled out for us the effect of radium on the skeleton and explained how it produced bone cancer. Has radium in any way, shape, or form affected the blood to the point of developing leukemia or arthritis?

Dr. LOONEY. As I mentioned, the hematological changes in these people have been minor. In one case the patient died from renal insufficiency and there was a question of leukemia, but I would have to look this up.

Representative VAN ZANDT. What about arthritis?

Dr. LOONEY. Arthritis—I suppose the effects of radium might be considered a destructive form of arthritis. These people get destruction of the hip so this is, in a sense, a form of destructive or degenerative arthritis.

Representative VAN ZANDT. Dr. Looney, to your knowledge, has radium produced any other effects to the human body other than bone cancer?

Dr. LOONEY. That I know of, no sir. The major effects are the skeletal changes, and the bone tumors.

Representative HOLIFIELD. Dr. Looney, your presentation has been very valuable, and particularly the well-documented written presentation, which will be included in toto in the record.

I appreciate particularly having the pictures of the bone structures that are represented by your clinical and experimental experience in the record.

Thank you very much.

(The prepared statement together with "A Study of the Dynamics of Strontium and Calcium Metabolism and Radioelement Removal" submitted by Dr. Looney follows:)

THE BASIS FOR THE PRESENT MAXIMUM PERMISSIBLE CONCENTRATION FOR RADIUM AND ITS RELATION TO THE MAXIMUM PERMISSIBLE CONCENTRATION FOR STRONTIUM 90

A statement perpared for the Joint Congressional Committee on Atomic Energy on the subject "The Nature of Radioactive Fallout and Its Effects on Man," June 4, 1957, by William B. Looney, M. D.,[1] the John Collins Warren Laboratories of the Huntington Memorial Hospital of Harvard University, Massachusetts General Hospital, Boston, Mass.

INTRODUCTION

Man is constantly confronted with toxic agents in his environment, which, if present in sufficient quantities, may produce temporary or lasting changes. By the accumulation of clinical and experimental information about the quantity of these toxic agents which produce minimal changes, safety measures are established. The present maximum permissible concentrations for radioelements in use today are based primarily on the results of clinical and investigative studies made over the past half century.

The principal sources of material for the study of the effects of internally deposited radioactive materials in man have been individuals who were employed in the luminous dial industry and persons who received radium for medical purposes. Radium salts had been given orally and intravenously for hypertension, arthritis, anemia, and other medical disorders from about 1915 to 1930. The painting of watch dials with luminous materials containing radium, mesothorium and radiothorium started in this country in about 1914. Adequate safety measures were not taken until about 1925–27, following the deaths from bone tumors, anemia, and crippling bone lesions which occurred in some of these workers (41).

The first report in regard to the maximum permissible concentration (MPC) for radium was made by the National Bureau of Standards in 1941 (9). Seven individuals, having from 0.02 and 0.5 micrograms of radium in their bodies from 7 to 25 years had no noted changes referable to the deposition of radioactive materials. However, death had occurred in patients having as little as 1.2 micrograms of radium. The maximum permissible level of body burden for the amount of radium which remains after early rapid elimination was considered to be 0.1 micrograms.

Two investigations of 50 radium patients and 28 luminous-dial workers were made in Boston and Chicago and recently reported (3, 25). As a result of these two investigations, greater reliance can be placed in the MPC for radium. These investigations have given information about the effects of radium deposited for long periods of time in quantities at or near the present MPC. One of the most important results of the recent investigations is that a relationship between an objective clinical finding (skeletal roentgenographic abnormalities) and the physical estimate of total body radium could be made within a specific dose range.

This report is a condensation of a review article published recently in the Journal of Bone and Joint Surgery (27), which summarizes the results of studies made on the radium patients and the luminous dial workers over the past 40 years.

The clinical course following the deposition of varying amounts of radium has been divided arbitrarily into two categories in this report.

A. Patients with total body radium content at or near the MPC of 0.1 micrograms with either no detectable clinical effects or minor skeletal changes

Thirteen of the seventy-eight patients studied in the Boston-Chicago investigations had between 0.01 and 0.4 micrograms of radium, and with the exception of 2 cases listed below, none had either symptoms or skeletal changes which

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could be attributed to radium. One patient with 0.4 micrograms of radium, age 83 years, was incapacitated with arthritis, therefore it was difficult to differentiate between the skeletal changes which may have been produced by radium from those which may have been produced by the skeletal disorder and disuse atrophy. Roentgenographs of the teeth of the other patient demonstrated areas of resorption in the teeth similar to those seen in other patients with greater amounts of radium. This patient was 32 at the time of examination. He had received radium water as a tonic between the ages of 8 to 10 years. The total body radium was only 0.15 micrograms of radium. It was considered that the early age of administration of radium may account for the dental changes with only a 50 percent larger radium content than the MPC of 0.1 micrograms.

B. Chronic effects of radium

1. Patients studied during the first and second decades following the initial medical and industrial use of radium (1915–35).—These patients were symptom free for about 10 years (31). Following the latent period, various changes relating to the skeleton began to develop. Death usually occurred from tumors of the skeleton, anemias or crippling bone lesions, or all three. Twenty-five of these cases were reported in the two decades which followed. Estimates of the radium content of the body began to be reported on these patients; however, the reliability of these estimates is hard to ascertain. These estimates ranged from about 5 to 100 micrograms of radium.

2. Patients studied during the third and fourth decades following the initial medical and industrial use of radium (1935–55).—Sixty-five of the seventy-eight patients evaluated in the recent Boston-Chicago investigations, having between 0.5 and 23 micrograms of retained radium, had either roentgenographic changes or symptoms, or both, which could reasonably be attributed to radium. The exceptions to this were three patients having betwen 0.5 and 1 microgram of radium having no skeletal abnormalities demonstrated roentgenographically. There was no proportionate increase in either the frequency or severity of the roentgenographic changes and symptoms with increasing amounts of radium in the body. The wide variation in the clinical abnormalities is consistent with the hypothesis that the changes observed are the result of a dynamic relationship between the reparative processes of the body and the destructive processes of the body acting in conjunction with the releterious effects of radium.

The mean age of the 28 luminous dial workers in the Boston-Chicago study was 21 years at the time of employment, and the mean age of the 50 patients at the time of radium administration was 34 years. The average time of retention was about 25 years in both groups, with a range of about 10 to 35 years. The average age of the patients in 1951 was about 50 years (range from 40 to 80 years). Seven of the 50 patients given radium have died; 5 died from tumors, 1 from anemia, and 1 from multiple causes; 11 of the 28 luminous dial workers are dead; 8 died from tumors, 1 from broncho-pneumonia, 1 from a urinary disorder, and another from renal insufficiency and leukemia. The average age at death for the radium patients was about 65 years, and the average age at death for the luminous dial workers was about 45 years. The average time of deposition of radium until death of the 18 patients was about 25 years, and average total body radium was about 5 micrograms.

As far as we have been able to determine, 10 of the 50 radium patients and 16 of the 28 luminous dial workers were discovered because of symptoms. It is apparent that these patients represent a biased sample from the population of radium patients and luminous dial workers. Correction for this biased sampling should be made in any attempt to relate the frequency of bone tumors and bone destruction in these patients with estimates of the frequency of these changes with comparable amounts of strontium 90.

RADIUM ELIMINATION

Radium is considered to be dispersed throughout the soft tissues and the skeleton when it first has been taken into the body (43). Most of the radium is eliminated during the first week (42, 43) (fig. 1). Fecal excretion is the major route of elimination in early and late cases. From 90 to 97 percent of the radium which is eliminated is eliminated in the feces and only 3 to 10 percent is eliminated in the urine (2, 35, 42). It has been reported that the rate of elimination following inhalation or ingestion is more rapid than that following intravenous administration (40, 42). Results from recent studies, however, indicate that these differences in the rate of elimination are not so pronounced as it was previously thought (26, 30, 35). Within a matter of

months, 90 to 99.9 percent of the radium is eliminated (40, 43). The age, the total amount of radium injected, and the estimates of the injected dose in 19 patients at 6 months, 12 months, and 20 years, have been reported (35). The average amount of the injected radium remaining at the times these estimates were made was 4.7 percent, 2.2 percent, and 0.8 percent, respectively.

The amount of the remaining radium eliminated daily becomes less and less (26). The available clinical data on the coefficient of elimination[2] of radium are given in figure 1. The marked change in the coefficient is elimination at 1 week has been considered to be the result of the elimination of the principal portion of radium from the soft tissues and the gastrointestinal tract and the change at 1 year has been considered to be the result of the elimination of the principal portion of radium from the more accessible parts of the skeleton. It is reasonable to conclude that the decreasing rate of elimination of radium is from the more inaccessible parts of the skeleton after 1 year.

About 15 to 35 percent of the absorbed radium is eliminated the first day. The amount of the remaining radium eliminated daily after 1 week is less than 1 percent. After about 10 years, it varies from 0.002 to 0.009 percent of the body content. The coefficient of elimination after about 20 years varies from about 0.002 to 0.016 percent of the body content (35, 43).

THE MANNER OF RADIUM DEPOSITION IN THE BODY

A. Autoradiographic study of radium deposition

Entire bones were sectioned serially and a comprehensive picture of distribution throughout the entire bone was obtained by placing the sections on roentgenographic film (3, 23). The precise manner of the deposition of radium was obtained by detailed autoradiography (1, 3, 23). Histological sections were covered with a photographic emulsion. After the emulsion and staining had been developed, simultaneous study of radium deposition and histopathological change was made.

Radium was found in small areas of high focal concentration irregularly distributed in both compact and cancellous bone. In compact bone only a small percentage of the Haversian systems and interstitial lamellae had appreciable concentrations of radium. In some instances radium was concentrated in 1 or 2 concentric lamellae, in other instances it was deposited around the central canal or periphery of the Haversian system.

The areas of radium concentration in traveeular bone were usually 5 to 15 micra in the greatest dimension. However, there was a wide range in size and shape of these areas, and they were found at any depth within the trabecula. In some instances linear concentrations ran parallel to the curvature of the trabecula for 50 to 100 micra. Heavy and rather uniform concentrations of radium were present at the junction of the articular cartilage and the traveculae of the long bones in the gross autoradiographs. In the study by gross autoradiography it was found that frequent small highly concentrated areas resulted in outlining the bone contours (fig. 8). Some cementing lines were clearly outlined by heavy concentrations of radium.

In some sections exposed for long periods of time there was a much less concentrated and a much more uniform distribution of radium. These findings are in agreement with existing theories that radium has more than one principal mode of deposition. The small highly concentrated areas may have been areas in which bone formation was taking place at the time of administration or redistribution. The more uniform and less dense distribution may be the result of inorganicion exchange (23). (See figs. 2, 3, 4, 5.)

HISTOPATHOLOGY

The histopathological changes which are most important diagnostically are: (1) the formation of an atypical osseous tissue in the trabecular spaces of cancellous bone, and (2) well differentiated areas of destruction in compact bone.

Martland divided the changes in the skeleton into three stages, all of which are primarily concerned with the bone marrow (31). In stage III, however, he did state that bone absorption and considerable decalcification did occur along with the replacement of bone marrow and noncellular fibroblastic tissue.

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[2] The coefficient of elimination is equal to the amount of radium eliminated per unit time (days) divided by the amount of radium in the body at the time the elimination measurements were made.

Studies of the skeletal changes produced in animals following the deposition of radioactive elements clarify the mechanism of production of the histopathological changes that have been found in human material. Heller and Bloom and Bloom concluded from their studies of such bone-seeking radioactive elements as strontium, yttrium, plutonium, and radium that similar changes were produced by all of these radioactive elements.

Following radium injections in mice the most spectacular change was a dense atypical bone in the metaphysis. This started from the proliferation of spindle cells which formed a dense fibrous tissue. Areas of calcification occurred in this fibrous tissue to form an atypical osseous tissue. It was also found to lesser degree in the shafts of the long bones and in the vertebrae. This was accompanied by the disappearance of osteoblasts, swelling and degeneration of cartilage cells, and death of most of the osteocytes. Repair began along with the destricutive effects of radium. In the lower dose ranges, reversible changes occurred so that in a matter of months normal bone was found on histological examination. Larger amounts of radium producted more severe and lasting changes. The epiphyseal cartilage varied tremendously in width, and the entire metaphysis was abnormal in appearance. The atypical osseous tissue remained in these animals at all intervals up to the termination of the experiment at 5 months.

The femoral shaft contained varying degrees of empty lacunae and dead oseocytes. The bones of the shaft varied from the relatively smooth contour to greater or lesser irregularities on the endosteal surface with projection into the marrow cavity.

Historical specimens have been obtained from 3 luminous-dial workers and 6 patients who had received radium. The radioactive elements had been present in the skelton from 12 to 35 years preceding biopsy and autopsy. Two luminous-dial workers and two patients who had received radium had bone tumors.

The histopathological changes were similar in certain respects to those changes found by Martland, Heller, and Bloom and Bloom (4). Atypical osseous tissue was present in cancellous bone. It was usually found near the articular surface of such bones as the humerus and femur, in addition to being found in the metaphyseal area. The atypical osseous tissue was laid down adjacent to the trabeculae in some areas. In some areas there was hyperplasia of the trabeculae, while in others destruction of the trabeculae was present. The trabecular spaces were usually filled with an acellular fibrous tissue. In general there was an absence of radioactivity in the atypical osseous tissue and accellular fibrous tissue was found in the autoradiographic study. In most cases, there was a prominent absence of osteocytes in the lacunae, and there was little evidence of bone regeneration.

In compact bone, the central canals showed a wide variation in size from normal to the nearly complete destruction of the entire Haversian system. A large number of the central canals were occluded with a dark staining material similar to the atypical osseous tissue in the trabecular spaces. There was usually an absence of cells in the lacunae. Minimal evidence of bone regeneration was observed, and areas of destruction usually were replaced with fibrous connective tissue.

In addition to the microscopic areas of destruction, macroscopic areas of destruction have been observed in compact bone 1 to 2 millimeters in width and 5 to 20 millimeters in length. Radium concentrations were rarely found in or around the macroscopic areas of destruction. Areas of transition in which both radium concentrations and microscopic change appear together have been found rather infrequently.

It has been shown that a correlation exists between the frequency of destructive changes and the amount of radium deposited in the body. It has been postulated that these macroscopic areas of destruction occur as the result of the fusion of adjacent central canals of Haversian systems undergoing destructive changes. It has been further postulated that the radium had been removed from the macroscopic areas of destruction by the time the changes occurred. If direct irradiation was the primary mode of production of the skeletal changes, more areas of transition should have been found in which both radium deposition and skeletal histopathological changes were present. It is evident that considerable difficulties are inherent in any attempt to reconstruct a pathological process which has been going on for 20 to 30 years from specimens taken at the termination of the process.

As a result of these observations, it is considered that radium deposited in the skeleton usually initiates a sequence of events which eventually produces patholog-

ical changes. These changes are probably the end result of many intermediate factors such as trauma, damage to blood supply, hormonal imbalance, decreased bone repair, and increased bone destruction from other causes. Evidence from the histological sections indicates that Haversian systems may undergo periods of resorption followed by periods of bone formation. There was a subnormal appearance of the bone in many instances. Some of the destructive changes may be the result of the inability of bone to maintain normal repair. It is evident that the relationship between radium deposition and skeletal change is complex. It is reasonable to conclude that when the destructive effects of radium and other deleterious intermediate factors become greater than the reparative processes of the skeleton, permanent alternations occurs (see figs. 6, 7, 8, 11, and 12).

HEMATOLOGICAL FINDINGS

In 1924, Castle, Drinker, and Drinker (8) reported the hematological findings of 22 luminous-dial workers. The erythrocyte count was below 4 million in 6 percent of these cases and above 6 million in 19 percent. The white-blood-cell count was below 7,000 in 27 percent of the workers. Abnormal erythrocytes occurred in 36 percent of the series. There was an increase in lymphocytes and monocytes and decrease in the polymorphonuclear neutrophils found on differential examination.

In 1943, the Public Health Service (41) made a study of 196 employees of luminous-dial-painting plants. Anisocytosis was found in 11 percent of these people, and poikilocytosis in 8 percent. The average erythrocyte count in the Public Health Service series was 4,300,000 as compared with an average of 4,500,000 in a series of 31 control patients.

Hematological data were available on four patients who died. Two of these patients died from malignant tumors—patient (R–24) and patient (B–7). Patient (R–44) died from aplastic anemia and patient (L–27) died from secondary infection and debilitation.

The marked pancytopenia that occurred in Martland's cases had not occurred in the patient evaluated in 1951. A short time before death, there were usually about 3 million erythrocytes per cubic millimeter and 10 grams of hemoglobin per 100 cubic centimeters. No hemorrhagic manifestations occurred in these patients. Some of Martland's cases had less than 1 million erythrocytes and less than 1,000 leukocytes per cubic millimeter shortly before death. He considered the anaemia which developed in his patients to be of a regenerative type resembling pernicious anemia, and he described the hematological changes in the following way: "The blood in this case showed a profound anemia, characterized by a large cell anisocytosis, by the presence of megaloblasts and by a marked leukopenia. There was not, however, a hyperbilirubinemia, and the van den Bergh tests were negative. The anemia is, therefore, not a hemolytic anemia. It is not an aplastic anemia, since there is marked embryonal blood formation. The fault lies in a long continued irritation of the hematopoietic system, the hemolytic or reticuloendothelial system being unaffected. There is a stage of stimulation followed later by sudden exhaustion of the erythroblastic and leukoblastic centers with the production of a rapid, fatal anemia with leukopenia, which fails to be influenced by any form of treatment." Red regenerative marrow was present in the femora of some of these patients.

These changes in the hematopoietic system are similar to those seen in the acute radiation syndrome. Most patients who lived less than 6 weeks following external radiation had hypoplastic marrow. Some patients who died at 4 to 5 months after exposure had diffuse myeloid hyperplasia which involved even such long bones as the femur. Occasionally the marrow appeared pink and gelatinous.

The bone marrow of patient (R–24) was studied in 1948, 18 years after radium administration. It was reported to be distinctly overactive. There was moderate erythroblastic activity with an excessive number of cells in mitosis without a shift to the left. The remainder of the cells were normal. The erythrocyte count was 5,300,000 per cubic millimeter and hemoglobin was 12 grams per 100 cubic centimeters at this time. The erythrocyte count was 3 million per cubic millimeter and hemoglobin was 9 grams per 100 cubic centimeters shortly before death in 1951. Small pink areas of hyperplasia was found in the femur and tibia at autopsy. Sternal marrow from patient (R–43) revealed extremely atrophic marrow shortly before her death in 1947. There were only small areas or erythropoiesis and the remainder of the marrow was composed principally of fat cells. No evidence of excess destruction, serious fat atrophy, or fibrosis

was observed. The hemoglobin at this time was 12.6 grams per 100 cubic centimeters and the erythrocyte count was 3,890,000 per cubic millimeter.

Patients who have over one microgram of retained radium are more likely to have anisocytosis, poikilocytosis, and hypochromia of the erythrocytes than those having under one microgram. However, significant hematological changes usually do not occur until late in the course of the disease.

In view of this additional information, it appears that little qualitative difference exists in the hematological response to internal or external radiation. If an individual had received large enough amounts of radioactive substances internally, hematological changes occurred which were similar to the hematological changes following exposure to large amounts of external radiation. In individuals who had received smaller amounts of either internal or external radiation, the hematological response at any given time was varied. The type and number of cells in the circulation at any specific time is the result of the natural survival of the cells and the balance between their radiosensitivity and their ability to recover from injury.

The red regenerative marrow that Martland described was in all probability an abnormal attempt of the hematopoietic system at increased production as a result of damage. Martland's term leukopenia anemia of regenerative type which was used to describe the hematological changes in the luminous-dial workers does not seem to be appropriate. The anemia that develops in these patients and the patients who had received radium would be more suitably placed in the category of primary refractory anemia in which either a hyperplastic or hypoplastic (aplastic anemia) bone marrow may be found. No leukemias have been found.

ROENTGENOGRAPHIC CHANGE

Areas of increased density have been found in cancellous bone on a skeletal roentgenographic examination. This roentgenographic change is the result of a typical osseous tissue in the trabecular spaces; infrequently it is the result of hyperplasia of trabeculae. Well-defined areas of decreased density were found in compact bone. These were the result of the destructive changes in the cortex of the bone.

These changes were divided into three groups, principally on a descriptive basis (24):

Group I: Areas of decreased density which were usually 1 to 2 millimeters in width and 5 to 20 millimeters in length in the long bones which gave a streaked appearance, and "punched-out" areas varying from 2 to 20 millimeters in the greatest dimensions present in the skull.

Group II: Areas of increased density usually associated with areas of decreased density with varying degrees of change in the trabecular pattern. These usually occurred in the femoral head, the humeral head, and the glenoid process, giving a mottled or motheaten appearance. There was an increase in frequency of biconcavities and collapse of the vertebras in patients having larger amounts of retained radium. Areas of increased density were found along the superior and inferior borders as well as small areas of increased density in the vertebral bodies.

Group III: The term "aseptic necrosis" has been used in referring to the changes in normal configuration. The heads of the femora, the bones of the feet, and the mandible were most commonly involved.

Serial roentgenograms of a few of these patients over long periods of time have been obtained. In most of the available serial roentgenographic studies, changes were not found until years after the deposition of the radioactive element. Periods of skeletal change may occur and may become stabilized or may improve. Later skeletal changes occur with increasing frequency.

For example, one patient (R-50) was given radium in 1922 (age 42 or 44). Ten years later skeletal roentgenographic changes characteristic of radium deposition occurred primarily in the mandible. These changes gradually improved. In 1940 (age 60) a significant increase in skeletal involvement occurred. There was a gradual increase in frequency and severity until her death in 1949. Roentgenograms of the skull of another patient (R-24) in 1948 demonstrated 12 areas of decreased density; the number of areas had increased to 22; however, there was minimal increase in the size of the areas. The size of these areas usually did not become greater than dimensions given in the description of the roentgenographic findings. Infrequently, however, they may be as great as 3 to 4 centimeters in diameter. The usual sequence of events was an increase in numbers, as well as an increase in the number of bones involved.

It is evident from these and other serial roentgenograms that these changes do not bcome detectable until many years after the deposition of radioactive elements. The possibility of skeletal histopathological changes being produced and remaining undetected reontgenographically for years must also be considered. There were no roentgenographic changes found on complete skeletal roentgenographic examination of patient (R-13) in 1950, with the exception of an aseptic necrosis of the right femoral head. However, minor histopathological changes characteristic of the deposition of radioactive elements were found in sections of the left fibula of this patient. In 1954, roentgenographic changes characteristic of the deposition of radioactive elements were present in the fibula and other long bones.

It has been shown that skeletal histopathological changes are reversible in animals following the deposition of radioactive elements. The history of another patient (R-50) demonstrates that skeletal changes as seen by roentgenogram are reversible in man following the deposition of radioactive elements. It is reasonable to assume, therefore, that reversible skeletal histopathological changes occur in man, as well as in animals, following the deposition of radioactive elements. When the reparative processes are able to be maintained at a level equal to the destructive processes, no detectable changes occur as seen by roentgenogram. If the ability to maintain skeletal repair is impaired or decreased, more and more permanent alterations probably result from the imbalance between the reparative and destructive processes.

Histopathological changes have been present to such a degree that roentgenographic changes have been found in many patients who are symptom-free. However, significant clinical changes usually do not occur in the absence of skeletal roentgenographic change. The results of skeletal roentgenographic, autoradiographic, and histopathological studies made on serial bone sections indicate that repeated skeletal roentgenographic examinations are the most satisfactory clinical methods for the early detection of skeletal alterations following the deposition of radioactive elements.

Thirty of the patients who were given radium for medical reasons and two luminous-dial workers were selected because each of these patients had had a complete skeletal roentgenographic examination. They were arranged in order of increasing amounts of retained radium from 0.5 to 14 micrograms, and the skeletal changes characteristic of the deposition of radioactive elements were tabulated. These patients were arbitrarily divided into three groups, those patients having between 0.5 and 1 microgram of retained radium, those having between 1.1 and 2 micrograms, and those having between 2.1 and 14 micrograms. Table I gives the age and sex of 32 patients 21 years after the deposition of radioactive materials.

Forty individuals were selected at random while undergoing physical examination at the Argonne National Laboratory for skeletal roentgenographic examination to act as a control group. It would have been desirable to have matched controls; however, the results of these roentgenographic examinations greatly minimized the possibility of the minor changes being present to any significant degree in the general population.

Five luminous-dial workers having between 0.02 and 0.4 micrograms of radium and 4 radium patients having between 0.01 and 0.4 of radium had complete skeletal surveys. One 83-year-old patient (0.4 micrograms of radium) had roentgenographic changes characteristic of radium deposition. The interpretation of these changes was difficult because of the marked arthritic abnormalities. Another patient with 0.15 micrograms of radium had areas of resorption of the teeth.

The five bones (skull, radius, ulna, tibia, and fibula) which were most frequently the sites of decreased density were first tabulated (chart I). The frequency of involvement was expressed as a percentage of the total number of bones that could possibly be involved. No distinction was made between unilateral or bilateral involvement, since the long bones were involved bilaterally in all but about 10 to 20 percent of the cases.

For example, in the 15 patients having between 0.5 and 1 microgram of radium, a total number of 75 bones could be involved; only 6 bones had changes characteristic of radium deposition or about 10 percent of the total number of bones. In group II, about 55 percent of the bones had characteristic changes, and in group III about 65 percent of the bones were involved.

Attempts were made to find some correlation between the severity of bone changes and increasing amounts of radium. The skeletal changes were divided into six grades of severity (27). With increasing amounts of retained radium

in the body, a proportional increase in roentgenographic changes does not occur. In addition, there is a marked individual variation in the amount of retained radium in the body. In some patients who had the greatest amounts of retained radium, only minor changes occurred, while other patients who had only one-fifteenth to one-thirtieth as much had more severe skeletal changes. Chart II graphically demonstrates this clinical observation in regard to the roentgenographic changes. Patients in group III had about 5 times as much radium as those in group II; however, there is only a 10 percent increase in the frequency of involvement. This lack of correlation between group II and group III is further emphasized by the fact that the average age of group III is 60 compared with an average age of 50 in group II. This finding and the finding of a significant increase in roentgenographic changes and symptoms at 1 microgram, even though the luminous-dial workers were 13 years younger, are suggestive that age at the time of deposition is not a major factor in the eventual production of roentgenographic changes. The fact that some of the patients had between 0.1 and 0.5 microgram of radium deposited for 25 to 30 years and had no roentgenographic changes is suggestive that time of retention per se may not be a major factor in the eventual production of roentgenographic changes. It is to be emphasized that these observations, suggesting that the time of deposition and the length of retention are not important, may well be the result of biased sampling of the patients, as well as an inadequate number of patients.

The most important result of these studies is that a definite correlation has been made between an objective clinical finding and the estimated amount of radioactive element retained in the body. For the first time, a semiquantitative relationship between the frequency of the roentgenographic changes and retained radium has been established within a certain dose range. No characteristic roentgenographic changes have been observed in patients having under 0.1 microgram of radium and relatively few changes have been found in patients having between 0.1 to 0.5 microgram of radium. Between 0.5 and 1 microgram of radium changes began to occur with increasing frequency and those patients having over 1 microgram of radium had a considerable increase in the frequency of the roentgenographic changes when compared with the patients having under 1 microgram of radium. (See figs. 9, 10, 11, 13.)

SYMPTOMS

Symptoms which can be reasonably attributed to radioactive element deposition usually result from destructive changes in the skeleton in patients with small amounts of radium. When symptoms do occur as a result of skeletal change, there are usually not severe in comparison with the extent of skeletal damage. In some instances, there is neither a progression of the skeletal lesions nor of the symptoms following the production of the skeletal change. For example, aseptic necrosis of the head of the femur in one patient developed in 1940 (age 30 years). This was followed by limping and discomfort on walking. However, during the period between the onset of symptoms and the examination in 1951, little progression of either limping or discomfort on walking occurred. There was also minimal discomfort following the aseptic necrosis of the head of the radius in another patient (R–23) since the beginning of symptoms in 1947. Another patient had aseptic necrosis of the head of the femur 22 years after the administration of radium at the age of 50 years (25, 27). She had no other symptoms or changes that could be related to the deposition of radioactive elements.

Skeletal changes usually occur in bones subject to weight-bearing or to repeated trauma (31). Aseptic necrosis usually occurs in the heads of the femora and the bones of the feet. Collapse of the veterbrae is occasionally found, while fractures almost always occur in the femoral shaft. One patient (L–27) fractured her left femur while pressing her foot to the floor of an automobile. Patients (L–14) and (R–35) sustained fractures of the shafts of the femur on minimal trauma. Healing of the fractures was delayed, but proper union occurred in all patients. Rarely, patients may have femoral fractures with permanent nonunion. Patient J. J. had collapse of some of the vertebral bodies with associated pain that required the wearing of a Taylor brace for control, while patient (R–44) had more marked vertebral changes with minimal discomfort.

Many of the patients did not have any symptoms which might be attributed to radium intake. Other than an increased risk of skeletal injury or tumor

formation, they may never have any recognized clinical changes as a result of radioactive element deposition.

In attempts to correlate clinical change with the amount of radium in the body, marked variations were found. The history of patient (R–49) illustrates this clinical observation. The only difficulty that he had was a march fracture of the foot which had healed uneventfully. The patient was asymptomatic at the time of examination in 1951 and only minor skeletal roentgenographic changes were present which could reasonably be attributed to the deposition of radioactive elements. Fourteen micrograms were present in his body, 15 to 30 times the amount present in some patients who had severe skeletal changes or tumor formation.

The time between the deposition of the radioactive elements and the onset of the first symptoms was analyzed in order to see whether any correlation could be made between this period and increasing amounts of retained radium. Twenty-one of the 25 luminous-dial workers had symptoms referable to radioactive-element deposition and 25 of the 50 patients who had received radium had symptoms that could reasonably be attributed to radioactive-element deposition.

The luminous-dial workers and patients who had received radium were divided into two groups. The first 10 patients of the luminous-dial workers who had symptoms and had between 0.1 to 1.5 micrograms of retained radium were compared with the 11 luminous-dial workers having between 1.6 and 18 micrograms of retained radium. The average time before symptoms occurred in the first group was 16 years and in the second group was 15 years. The 25 patients who had received radium who had symptoms referable to the deposition of radioactive elements were divided into 2 groups. The first 12 patients had between 0.7 and 4.2 micrograms of retained radium, the other 13 patients had between 5 and 22 micrograms. The average time before symptoms occurred in the first group was 17 years and in the second group was 16 years. There was no increase in the time interval before symptoms occurred in the first group, even though there was from about 10 to 100 times less radium in the body. It should be noted that the period of latency before the onset of symptoms that could reasonably be attributed to the deposition of radioactive elements varied from 1 to 32 years.

Since no relation between increasing amounts of radium and the time before symptoms occurred from deposition of radioactive elements was found, the frequency of involvement of the femur was examined to see if some correlation could be made with the retained radium. Nine patients who had received radium had aseptic necrosis of the head of one or both femora as did luminous-dial workers.

It is well known that aseptic necrosis of the femoral head usually occurs as a result of trauma or following fractures of the neck of the femur. It is also true that aseptic necrosis does occur in which the etiology cannot definitely be established. In these cases, it is considered that circulatory disturbances are the primary cause of the necrosis. The cause of the disturbance is poorly understood; it may be from gradual occlusion or from trauma (37). Unfortunately there is little information concerning the incidence of unexplained aseptic necrosis of the femoral head. Estimates of the occurrence of aseptic necrosis of the femoral head from all causes approximated 1 in 200 in an orthopedic practice.

The 14 percent incidence of aseptic necrosis of the femoral head in these patients seems unusually high in the absence of a history of antecedents trauma or fracture. It is interesting to note that all of the lesions occurred in patients having 0.7 microgram or more of radium in their bodies. The average time from the deposition of the radioactive elements until symptoms referable to the femoral head occurred was 15 years, approximately the same as that for symptoms in all other parts of the skeleton. The time of onset varied from 9 to 22 years.

Almost all of the fractures occurred in the shaft of the femur. Some of the patients fractured both femora, as well as sustaining refracture one or more times. Fractures following such minimal trauma as pressure of the foot on the floor of the car emphasize the fragility of the skeleton that may result from the deposition of radioactive elements.

Fractures of the femur occurred in 4 of the luminous-dial workers and in only 1 patient who had received radium. Again it should be noted that all of the fractures occurred in patients having 0.9 microgram or more of retained radium.

TUMOR FORMATION

The clinical course of patients in whom malignant tumors eventually develop is illustrated in the following case histories.

Patient (L–21) began to have symptoms as a result of skeletal changes 11 years after employment as a luminous-dial worker in 1934 (27). Several skeletal lesions occurred over a period of years following the deposition of the radioactive element. A biopsy specimen was taken from the left femur in 1950, following the clinical diagnosis of chronic osteomyelitis. The histological diagnosis at that time was a low-grade inactive fibrous osteomyelitis associated with recent slow-growing cancerous type of fibrous osteoma. In 1952 pain began to occur and to increase in frequency in the left femur, and the roentgenograms were interpreted as being suggestive of malignant changes in the medial condyle. The extremity was amputated and an osteogenic sarcoma was found throughout the femur on histological examination. The patient died a few months later.

Patient (R–24) had noted the onset of pain in the left foot 18 years (1948) after the administration of radium water (27). Roentgenograms of the foot a few months later revealed an area of decreased density in the left tarsal navicular. The condition became so severe that it necessitated the use of a cane. Roentgenograms at that time revealed marked destructive changes of the tarsal bones. Because of the marked increase in symptoms and the destructive changes in the foot in 1950, a biopsy was done and fibrosarcoma was diagnosed on histological examination of the specimen. The pain became generalized and required more and more analgesics to control; it was described as boring and burning in character. Intermittently, sharp knifelike pains occurred which lasted for a few seconds. The pain was not significantly influenced by motion, position, and temperature. The leg was amputated below the knee in February 1951. Examination of the amputated tibia revealed extension of the tumor. A sarcomatous lesion developed on the end of the stump a few weeks after the operation. The patient died in August 1951, as a result of generalized metastases. Areas of fibrosarcoma were found throughout the skeleton. In some parts of cancerous bone, it was difficult to distinguish between the fibrosarcoma and the fibrous connective tissue found in the trabecular spaces of patients without malignant involvement.

It is evident from the case histories of patients (L–21) and (R–18) that the transition from a benign to a malignant lesion may be similar to that in many skeletal diseases. Both of these patients were first considered to have osteomyelitis and then osteoid osteoma. In patient (R–17) the diagnosis of osteoid osteoma of the finger had been made 1 year prior to the diagnosis of osteogenic sarcoma. Characteristic histopathological changes due to the disposition of radioactive elements were found in reviewing the specimens from which these diagnoses were made.

The history of patient (L–8) emphasizes the need to follow these patients carefully. Patient has led a relatively normal life since 1934 as a result of prompt attention to symptoms occurring in the elbow. Following a biopsy and histological diagnosis of osteogenic sarcoma, the arm was disarticulated at the shoulder.

Figure 14 shows the sites of origin of the tumors that developed in 13 of the 78 patients. Eight of these patients were luminous-dial workers and five were given radium for medical purposes. Table II gives the age at the time of administration, the time from administration until the first symptoms occurred which could reasonably be attributed to the deposition of radioactive elements, the time from the first symptom until death, and the amount of retained radium. Since both groups of patients have approximately the same amount of radium retained in the body for about the same length of time it is not possible to make any differentiation between luminous-dial workers and patients who were given radium in relation to the estimated radium present. The average age at the time of employment in the luminous-dial workers was 17 years as contrasted with 39 years as the average age at the time of the administration of radium.

Most of the patients in whom malignant tumors eventually develop follow a similar course. There is a latent period followed by symptoms which usually developed from changes in weight-bearing bones and bones which are subject to repeated trauma. Later tumor formation occurs at a site which is usually not the site of the initial symptoms. When the malignant tumor develops, there may be a marked acceleration of the disease process and death usually occurs about 1 to 4 years after the symptoms began at the site where the tumor originated.

THE RELATION OF THE CLINICAL CHANGES TO THE AMOUNT OF RADIUM PRESENT IN THE SKELETON

A. Hematological changes

Minor changes occurred with greater frequency in the erythrocytes in patients having more than 1 microgram of radium than those patients having less than 1 microgram. The marked anemias found in the earlier cases were not present in the 78 cases studied recently. Two patients developed anemias a short time before death. Only one leukemia has been reported.

B. Roentgenographic changes

One of the most important results of the recent Boston-Chicago investigations was the ability to correlate the frequency of occurrence of roentgenographic changes characteristic of radium deposition with the physical estimates of total body radium. Thirty-two patients[3] having complete skeletal roentgenographic examinations were arranged in order of increasing amounts of radium and the changes tabulated (review section on roentgenographic changes.)

C. Symptoms

Symptoms[4] which can reasonably be attributed to radium occur as a result of changes in the skeleton. Symptoms which could reasonably be attributed to radium did not occur in any of the 78 patients who had under 0.4 microgram of radium. Nine patients who received radium medically and two luminous-dial workers developed destruction of the femoral head. The average time for symptoms to occur was 15 years (range 9–22 years). All of the patients who had aseptic necrosis of the femoral head had 0.7 or more micrograms of radium in the body. The 5 patients who had fractures of the shaft of the femur had 0.9 microgram or more of radium in the body.

The results of these investigations indicate that the relation between the destructive effects of the radioactive elements remaining in the body for long periods of time and the clinical changes produced, is a dynamic relationship between the destructive and reparative processes of the body. For example, some of the patients had aseptic necrosis of the femoral head with less than 1 microgram of radium, while others, with 10 micrograms or more, had no deleterious effects. It is reasonable to assume that these patients had other disturbances in the femoral head, such as an unsatisfactory blood supply. Another example of this dynamic relationship was demonstrated in the patient who had increased changes during pregnancy which subsided with the delivery of the child. Still another possibility is that certain periods of increased bone destruction or reduced bone formation may occur from disease processes which later revert to normal. One patient had rather severe changes in the mandible which later subsided.

D. Bone tumors

The 15 malignant tumors which developed in the 78 patients recently evaluated were found in individuals containing for 0.5 to 10 micrograms of radium in their bodies.[5] The patient having 0.5 microgram of radium was a luminous-dial worker. (It is reasonable to assume this patient ingested mesothorium and radiothorium.) The patient with the lowest radium concentration, who had received radium medically, had 0.9 microgram of radium. The patient with the lowest concentration of radium, which was considered not to be contaminated with members of the thorium decay series, and who developed a bone tumor, had 3.6 micrograms of retained radium. The accumulated radiation dose with 3.6 micrograms of radium was estimated to be about 5,000 rads during the 25 years from radium administration until tumor formation.[6]

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[3] One patient which was evaluated later with 0.15 microgram of radium had dental changes.

[4] Symptoms is a term used to denote subjective awareness of an abnormality. When skeletal changes progress to such a degree that destruction of the hip or fractures occur—symptoms are noted by the patient.

[5] See fig. 14.

[6] The estimates of continuous radiation dose is based on the assumption that 0.1 microgram of total body radium will deliver a dose of 3 rads per year to the skeleton. It was found in the radium excretion studies made in the Chicago investigations that the retention of radium could be expressed as an approximate power function of time (27). From this information Brues and Tyler derived an expression for the estimation of the cumulative radium dose from the estimation of the instantaneous dose rate at the time of measurement. Based on the equation of Brues and Tyler, the total cumulative dose would be approximately twice the dose estimated at the time of measurement.

Only 2 bone tumors have developed in the 52 of the 78 patients who were not discovered because of symptoms arising from the skeleton. The frequency of bone tumors in this group is 2 percent, in contrast to a frequency of 14 percent for the entire group of 78 patients.

Vaughan (45) (1956) reported 39 cases of sarcoma arising in bone following external radiation have been recorded in the literature. The latent period between receiving radiation and the development of the tumor was 3 to 11 years. The radiation dose was not known in all of these cases; however, in most recorded cases it was estimated to be usually greater than 3,000 roentgen (1,500–7,000 roentgen change).

Cruz et al (10) (1957) reported an additional series of 11 cases in which the sarcoma of bone occurred from 4 to 24 years after external radiation. The total radiation dose given ranged from about 1,000 roentgens to 5,000 roentgens, and was given over a period varying from 1 month to 9 years.

COMMENTS ON THE POSSIBILITY OF BONE TUMOR FORMATION FROM STRONTIUM 90

The assumption is often made that the incidence of the effect of strontium 90 is proportional to the magnitude of the dose. This assumption has been used to estimate the bone tumors which may be produced from the low concentrations of strontium 90 in the skeleton from fallout. There is no exact evidence either proving or disproving this assumption, but there is some clinical evidence which suggests that this assumption is over cautious.

The 50 bone tumors which have been known to have been produced in man, and reported, were summarized in the previous section. The skeleton in the localized area of tumor induction received at least 1,000 roentgens, and usually more than 3,000 roentgens of radiation. If it is assumed that the radiation dose to bone is greater by a factor of 2 than the measured skin dose, then the minimum observed carcinogenic dose from external radiation would be about 2,000 rads, with the majority of the tumors being produced by more than 6,000 rads of external radiation.

The patient with the smallest total body radium known to induce tumor formation, in which the possibility of contamination of the thorium series in unknown, died from a bone tumor in 1952. The estimated total body radium was 0.9 microgram. The time after administration is unknown, however, it is reasonable to assume that it was about 25 years. Based on the estimates above, the patient would have received a total accumulated dose of about 1,800 rads during the 25-year period.

Histopathological changes have been demonstrated by roentgenographic examination of the skeletons of radium patients prior to development of the tumors. It is generally considered that the bone tumors develop in or around areas of atypical osseous tissue formation. Bone tumors have been shown to develop in or around the abnormal bone formation in animals given plutonium (22). If it could be shown that these histopathological changes are preliminary steps to bone tumor formation, then it could be assumed that as long as the body reparative processes prevented the abnormal bone formation, bone tumors would not develop. It should be emphasized that the associated histopathological changes seen prior to bone tumor formation in the radium patients may be coincidental findings. Proof of a correlation must await a better understanding of tumor induction by radiation.

In reviewing the latent period for tumor induction in the luminous-dial workers reported by Martland in 1931 (31), it was found that the latent period for tumor formation was about 5 to 10 years in 6 patients in this study who developed tumors. Only 3 of these patients had estimates of total body radium reported and these estimates were 6, 15 and 50 micrograms. (See table II.)

The eight luminous-dial workers who were examined in the recent Boston-Chicago investigations, and who died from bone tumors, lived for an average of 25 years after beginning employment. The average total body radium was 3.4 micrograms (range of 0.5 to 10 micrograms).

It is considered that little reliance can be placed in estimating the minimum total body content for tumor induction over a life span from this meager data. However, if we do estimate the total body radium which would have a latent period for tumor induction greater than the average life expectancy (80 years)

from the 2 average radiation doses and latent periods given above, it would be 0.3 and 0.4 micrograms of radium respectively.[7]

If we assume that 0.5 micrograms of radium retained for 70 years would be the minimal body burden necessary to cause skeletal sarcoma, this would mean that a cumulative radiation dose of about 2,000 rads would be necessary to induce tumor formation. It is considered that the most reliable estimates of the radiation dose necessary to induce bone tumors over a life span of 70 years, should be equivalent to the total radiation dose necessary to induce carcinogenesis in 5 to 30 years (2,000 rads). Therefore, the estimates of the total radiation dose from strontium 90 necessary to cause bone tumor formation in man during a life span of 70 years will be based on the best available estimates from human data of about 2,000 rads. It may be calculated that 1×10^{-9} curies of strontium 90 will give a radiation dose to the skeleton of 3 millirads per year, or 0.2 rads in 70 years under equilibrium conditions. Based on the preceding assumptions, the total skeletal radiation dose from 1×10^{-9} curies of strontium 90 over a 70-year period would be in the order of one ten-thousandths total skeletal radiation dose known to produce tumors in man, either from external or internal radiation.

COMMENTS ON THE PRESENT MPC OF RADIUM

Results of the Boston-Chicago investigations have shown that clinical changes begin to occur in patients with 0.4 micrograms of radium. The fact that changes are found to occur in man with 4 times the present MPC of 0.1 microgram of radium instead of 12 times the MPC when it was established in 1941, is offset by the greater confidence in the MPC by the studies of a much larger group of patients with radium present in their bodies for longer periods of time.

It is reassuring that it has not yet been necessary to change the MPC of radium of 0.1 micrograms following the large amount of information that has accumulated since it was first established. There are certain considerations, which, if sufficient information becomes available, may permit raising, or may necessitate lowering, the present MPC. These are as follows:

A. Factors which may permit the raising of the MPC for radium

1. The contribution of the thorium series to the dose from radium.—It is well established that mesothorium I (half life 6.7 years) and radiothorium (half life 1.9 years) were used in the luminous-dial paints, and mesothorium has been found in patients given radium medically. The radiation dose from mesothorium in some of these patients has been significant. It has been the major source of the cumulative radiation dose in some of the luminous-dial workers.

The time relation in this question is very important. It is assumed that reversible changes are produced by the presence of mesothorium, then its major clinical effect should have occurred before the changes began to occur in these patients. Roentgenographic changes do not begin to occur until 15 to 30 years after the deposition of the radioactive elements. However, once changes begin to develop in these patients, there is usually a gradual increase in the frequency and severity rather than a decrease. If the changes produced during the period immediately after deposition are irreversible and contribute significantly to the changes that are being seen in these patients 20 to 30 years later, then it may be possible to elevate the MPC. Further work is in progress in an attempt to clarify the relationship of mesothorium, radiothorium, and radium in the clinical changes observed.

2. Controls.—Even though the roentgenographic changes found in these patients are characteristic of radium deposition, further control studies to estimate more reliably the incidence of these changes in the general population should be carried out. The skeletal roentgenographic findings may eventually prove to be the most reliable method for the early detection of clinical changes from the deposition of bone-seeking radioelements. A better definition of the normal limits of skeletal variation is needed, in order that a more precise evaluation of the skeletal changes in the radium patients may be made.

The destruction of the head of the femur is another clinical finding in these patients which emphasized the need for further control studies; 11 of the 78 patients had destruction of the hip. This frequency of aseptic necrosis is

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[7] These estimates are consistent with the findings of animal data of Brues[6], in which the latent period was found to vary inversely with the square root of the dose. The value of 0.3 micrograms of radium is obtained from assuming that the latent period of 10 years is increased by a factor of 8, and the average radium content is 25 micrograms. The value of 0.4 micrograms is obtained from assuming that the 25 years' latent period is increased by a factor of 3, and the average radium content of the patient is 3.4 micrograms.

unusually high in the absence of a history of antecedent trauma. Proper evaluation of this condition in the radium patients must await a better understanding of the cause of aseptic necrosis, as well as more reliable information as to its occurrence in the general population.

B. Factors which may necessitate lowering the MPC of radium

1. Age at the time of administration and duration of retention of radium.—The average age of the 50 patients at the time they received radium medically was 34 years. The average age of the luminous-dial worker at the time of employment was 17 years. Almost all of these patients either received radium, or were exposed to radium from 1920 to 1930. It is evident that the clinical information on the effects of radium deposited in the body for more than 40 years is lacking. Since the findings of the recent investigations suggest that clinical changes are the result of an imbalance between reparative and destructive processes of the body, it may be found that small amounts of radium in persons of the older age groups may produce significant clinical changes. This may result from either diminished reparative abilities, or an increase in skeletal destruction from other debilitating diseases of the older age groups.

There are only a few patients available for study of the long-term effects of radium who received the radium before the age of 15 to 20 years. One of these patients had the smallest amount of total body radium in which dental change characteristic of radium deposition has been observed.

The problem of bone tumor production in the younger age groups is probably the most important question in this regard. The greatest incidence of osteogenic sarcoma is considered to occur in the second and third decades, and almost no clinical information is available on the effects of skeletal radium deposition before this increase in the incidence of bone sarcoma occurs. It is possible that the increased susceptibility to skeletal tumor induction might necessitate a significant lowering of the MPC for children.

2. Undected subclinical effects.—The present philosophy for the establishment of the MPC is based on the correlation of physical estimates of the quantity of the radioelement in the body with detectable clinical changes or abnormalities in clinical tests. As more refined tests are developed to detect abnormalities in body function, it may be discovered that the present MPC is producing changes which are not detected by present methods of clinical evaluation.

Another important consideration is that subclinical changes may cause a reduction in the reserve function of organs. This may go undetected in most instances. However, the combined effects of an intercurrent disease and the reduced function from the effects of radiation may cause more severe effects than either the disease or the radiation separately.

COMMENTS ON THE PRESENT MPC OF STRONTIUM 90 BASED ON THE INFORMATION GAINED FROM THE EFFECTS OF RADIUM IN MAN

The present estimates of the MPC of strontium 90 are based on the assumption that both radium and strontium are distributed in a similar manner in bone. It has been shown that the radium concentration in different parts of the skeletons of the radium patients and luminous-dial workers may vary by a factor of 10 and probably greater. The present MPC for strontium 90 is based on the comparison of the average skeletal does of strontium and radium. Since under equilibrium conditions the strontium 90 distribution would tend to approach uniformity, comparison of the maximum skeletal dose in the localized areas of high radium concentration in the radium patients and the maximum skeletal dose in the strontium 90 patients might permit raising of the present MPC of strontium 90 by as much as a factor of 10.

The best available estimates indicate that biological effectiveness of radium is 1 to 4 times that of strontium 90 (i. e., the radium RBE[8] = 1 to 4) when compared on an equivalent energy basis. This work is based on the results of animal experimentation (15, 16). Comparison of the radiation dose necessary to produce bone tumors in man from internal and external radiation indicates that the RBE of radium may be somewhat nearer to unity. However, the estimation of radiation dose from external radiation might be in error by as much as a factor of 2. If it can be established that the RBE of radium is greater than unity then it might be possible to increase the MPC of strontium 90 by as much as a factor of 4.

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[8] Relative biological effectiveness: The ratio of gamma of X-ray dose to the dose that is required to produce the same biological effect by the radiation in question.

It is considered that the best estimates that can be made in regard to the effects of strontium 90 over a life span (70 years) on the present incomplete information of the effects of radium in man are as follows:

1. The skeletal content of strontium 90 necessary to produce a bone tumor in a life span of 70 years would be in the order of 10 microcuries (100 times the present MPC of 0.1 microcurie of strontium 90 for the general population).
2. The skeletal content of strontium 90 necessary to produce significant changes, such as destruction of the hip, would also be in the order of 10 microcuries (100 times the present MPC of strontium 90).
3. The skeletal content of strontium 90 necessary to produce minimal skeletal changes which could be demonstrated roentgenographically, would be in the order of 2 microcuries (20 times the present MPC of strontium 90).

It should be emphasized that these estimates of the concentrations of strontium 90 which may produce skeletal damage are the result of estimates based on the best available information at present.

TABLE I.—*Age and sex distribution of 32 patients having between 0.5 and 14 micrograms of radium present in their bodies on an average of 21 years after deposition*

	Group I (0.5 to 1 microgram)	Group II (1 to 2 micrograms)	Group III (2 to 14 micrograms)	Total
Sex:				
Male	4	2	5	11
Female	11	6	4	21
Total	15	8	9	32
Age:				
30 to 40	1	0	0	1
40 to 50	9	4	2	15
50 to 60	3	3	2	8
60 to 70	0	0	3	3
70 to 80	2	1	2	5
Average age (years)	49	51.9	60.9	----------
Average time (years) since administration	20.3	22.2	22	----------

CHART I

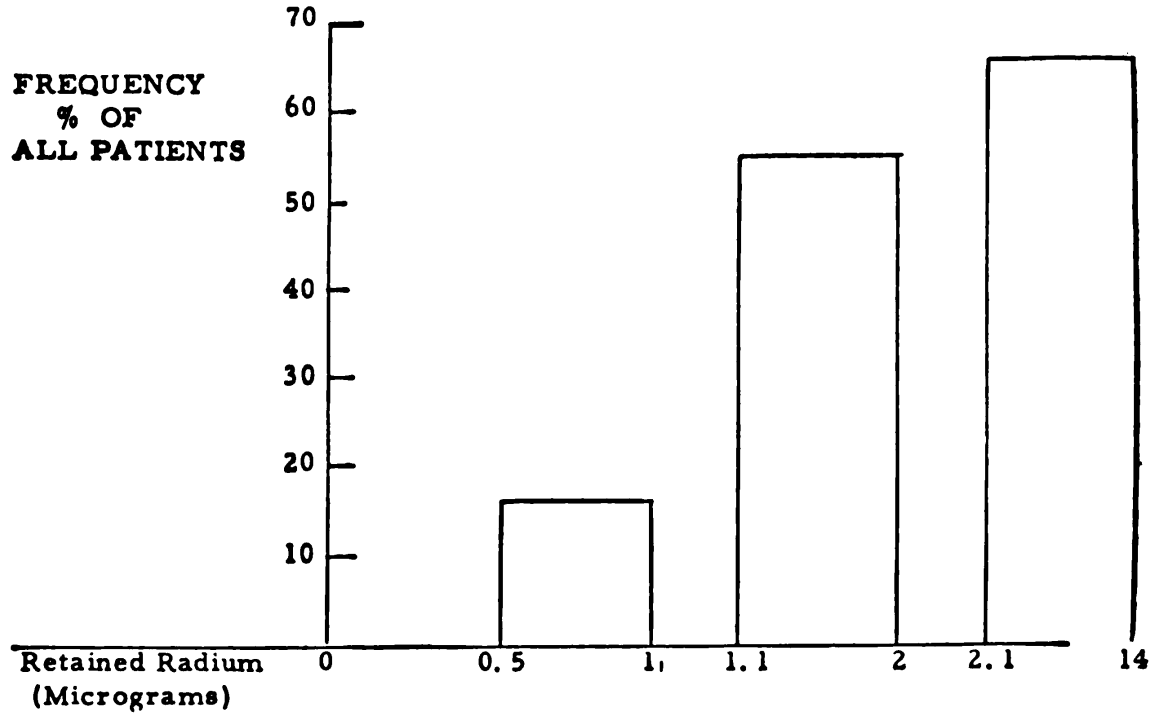

TABLE II.—*Time interval between deposition of radio-elements and occurrence of symptoms, tumor formation, and death in the 1931 and 1951 investigations*

	Number of patients	Average age at administration (years)	Average time from administration to death (years)	Average retained radium (micrograms)
Luminous-dial patients	3	17	10	23
1931 series				(6–50)
Range, years		16–21		
Luminous-dial patients	8	17	26	3.4
1951 series				(.5–10)
Range, years		15–21		
Radium patients		39	26	3.1
1951 series	5			(.8–8)
Range, years		25–49	1–8	

FIGURE 1

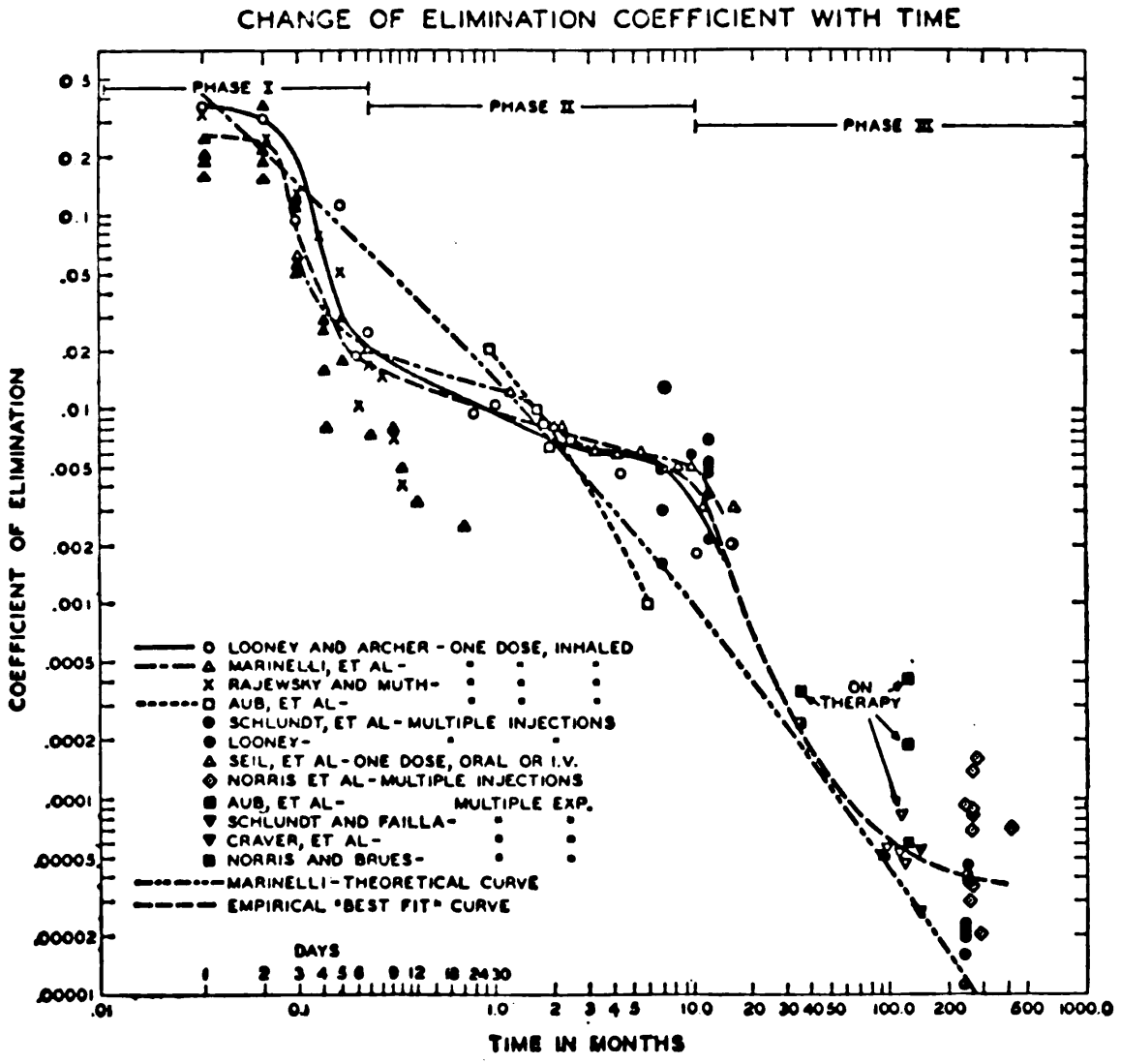

FIGURE 2.—Gross autoradiogram of a longitudinal section of the femur of patient M. K. Note the irregular distribution of radium in the cancellous bone of the upper end of the femur and the small areas of focal concentration in the cortex. The contour of the head is outlined by small concentrations at the junction of the articular cartilage and cancellous bone. The medial portion of the shaft is outlined by the small highly concentrated areas near the periosteum and the endosteum. (Reproduced courtesy of Journal of Bone and Joint Surgery.)

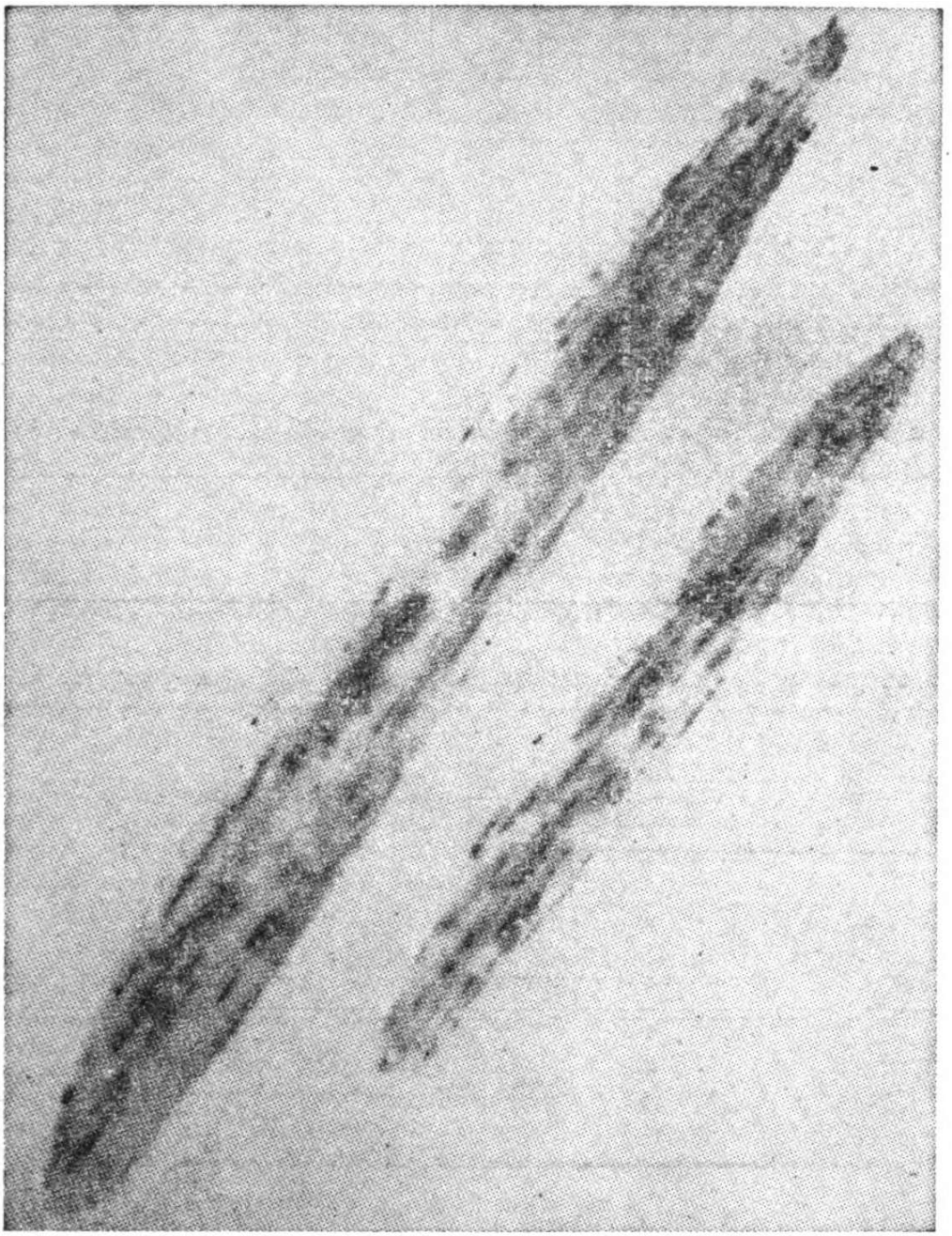

FIGURE 3.—Gross autoradiograms of two longitudinal sections of the cortex of a femur from patient R–24 (5-week exposure). Smaller section was taken 2 millimeters from the periosteum and the larger section was cut just medial to it. Areas of concentration are more frequent in the smaller section and the lower end of the larger section, which are nearer periosteum. The large area of darkening in the center of the larger section is the result, probably, of an endosteal concentration of radium, since this section was on the periphery of the narrow cavity. (Reproduced courtesy of American Medical Association, Archives of Pathology.)

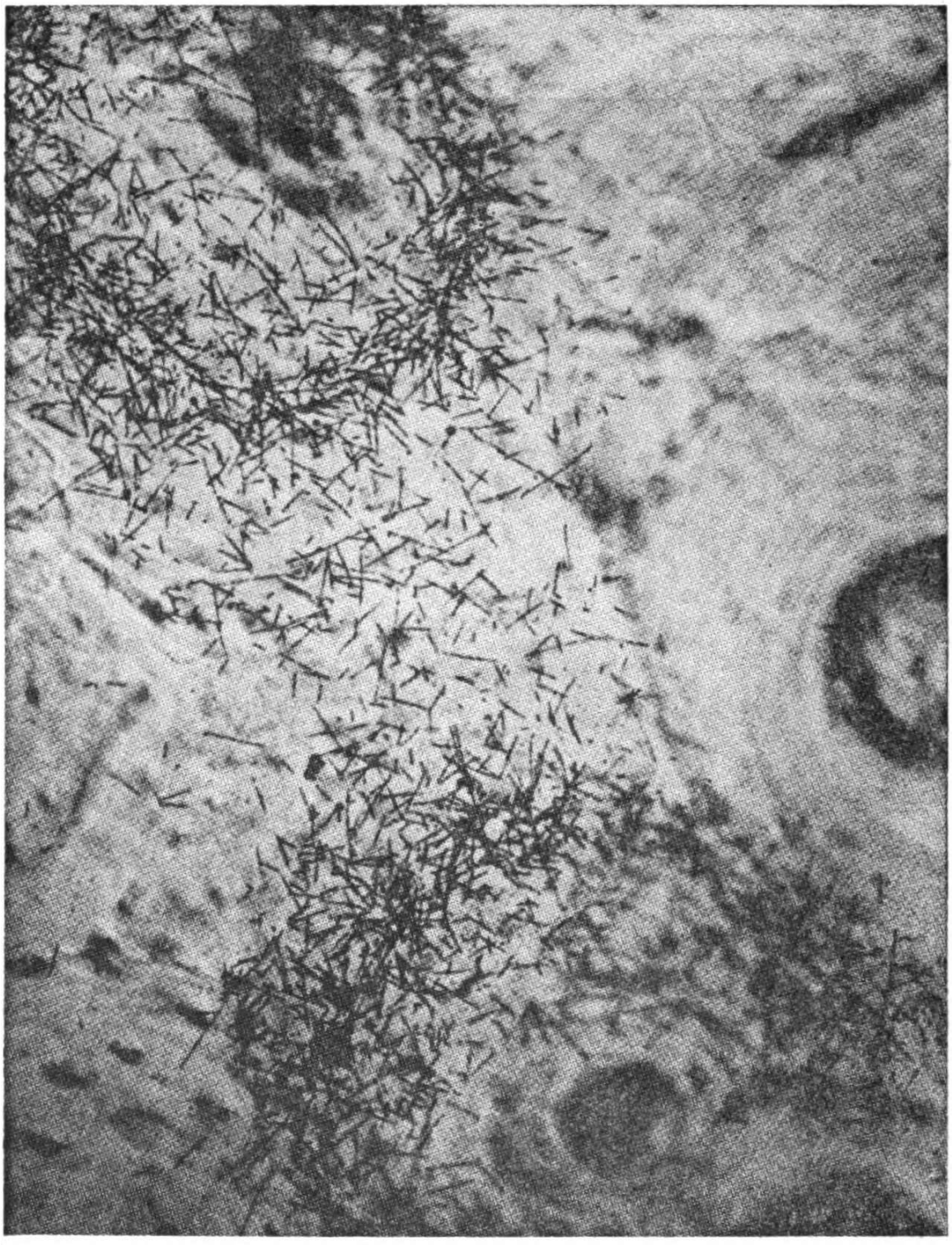

FIGURE 4.—Detailed autoradiogram of a cross section of cortical bone from the humeral shaft of patient R-24 (5-week exposure). Two of the small number of Haversian systems have radium concentrated in this section. The greatest concentration of radium is on two concentric lamellae in the center of the Haversian system. Alpha tracks are less dense in remainder of the two Haversian systems and interstitial lamellae between the systems. Observe that the activity suddenly falls off around these areas of concentration and that the rest of the photomicrograph is free of alpha tracks (X 207). (Reproduced courtesy of American Medical Association, Archives of Pathology.)

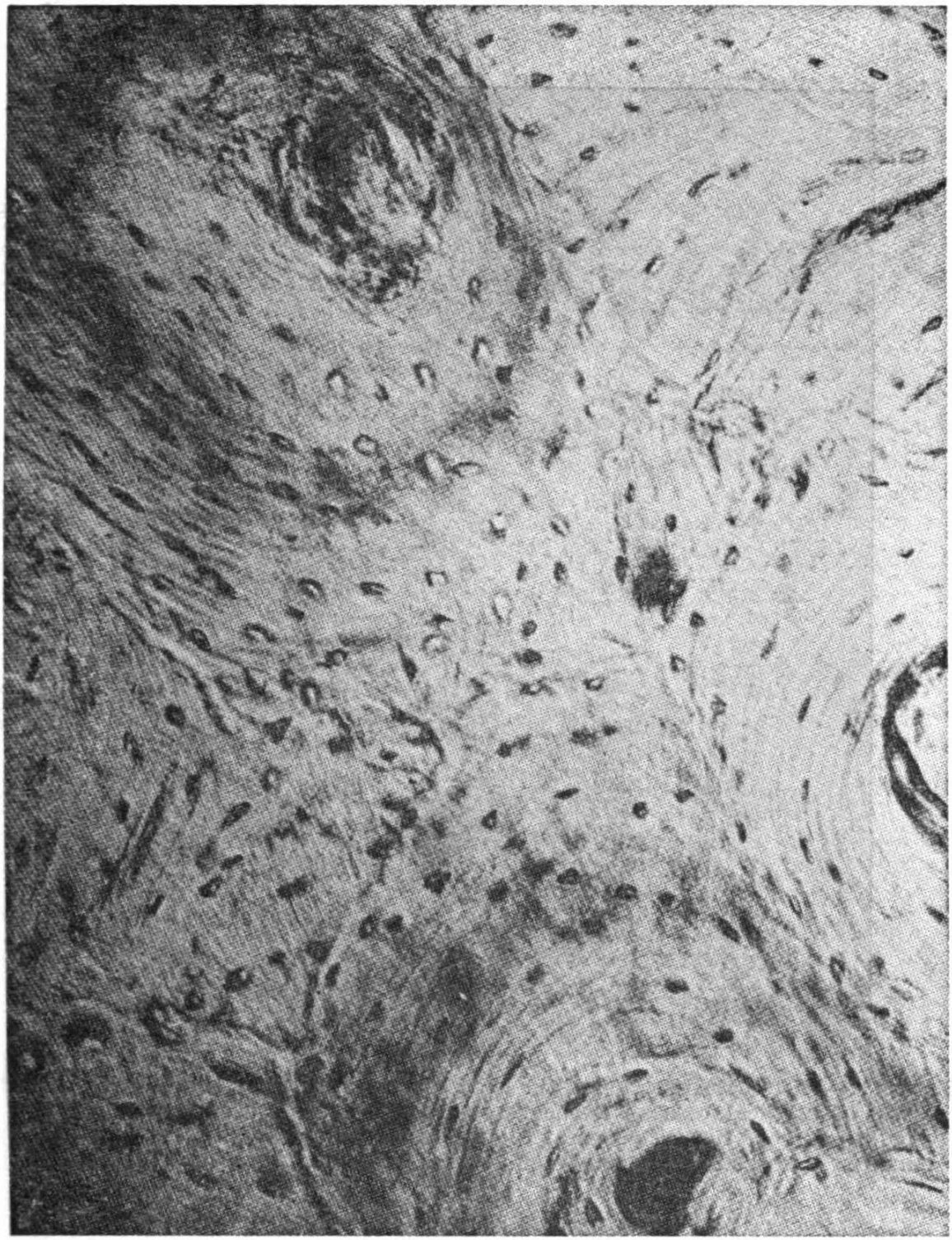

FIGURE 5.—Photomicrograph of bone underlying detailed autoradiogram shown in Figure 4. Note the dark concentric rings outlining lamellae with greater concentration of radium. Central part of Haversian system in upper left is undergoing destructive changes. This, together with figure 4, demonstrates how radium concentration and histopathological changes can be studied at the same time (X 218). (Reproduced courtesy of American Medical Association, Archives of Pathology.)

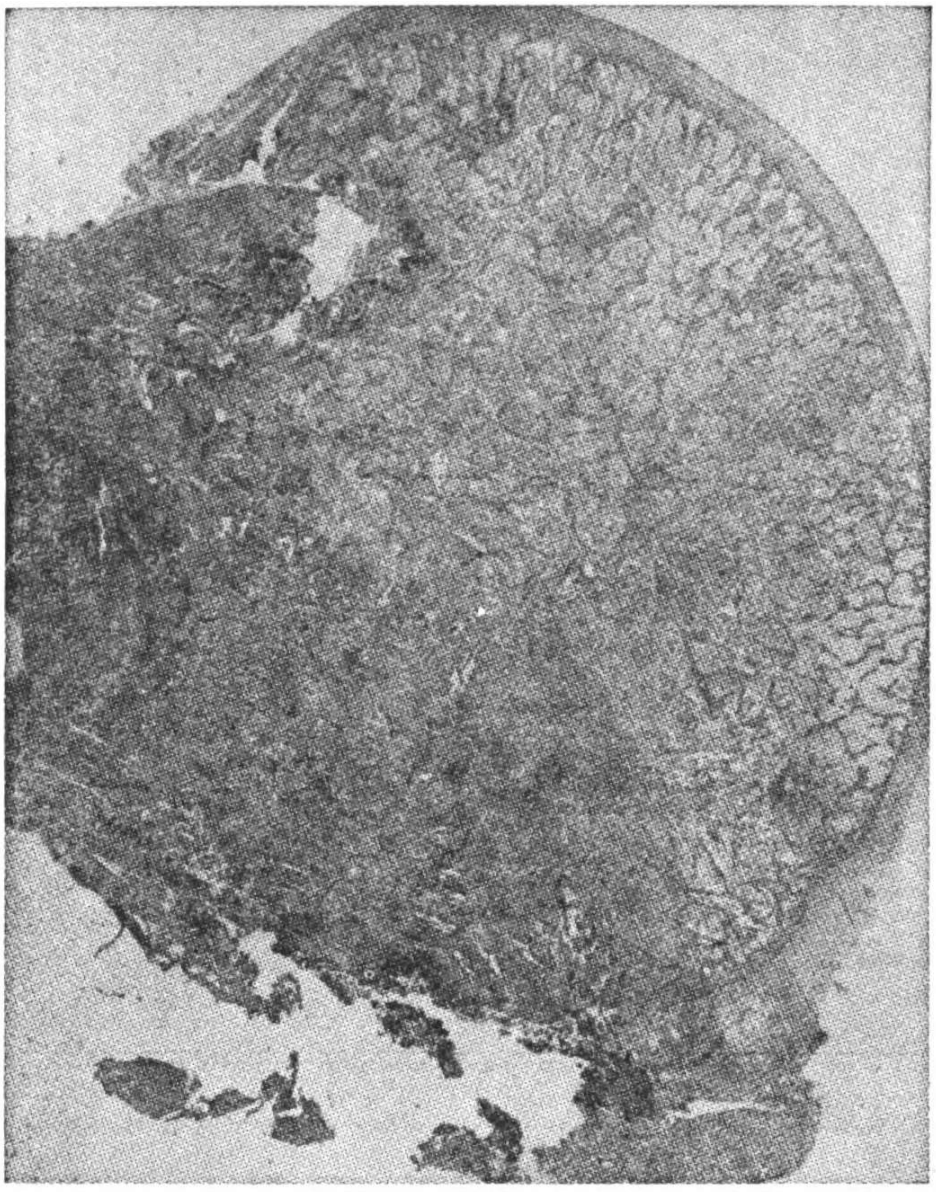

FIGURE 6.—Histological section of the head of a right humerus (fig. 2). Note the areas of atypical osseous tissue throughout the head. The largest area of atypical osseous tissue is seen at the top of the photograph near the articular surface. (Reproduced courtesy of Journal of Bone and Joint Surgery.)

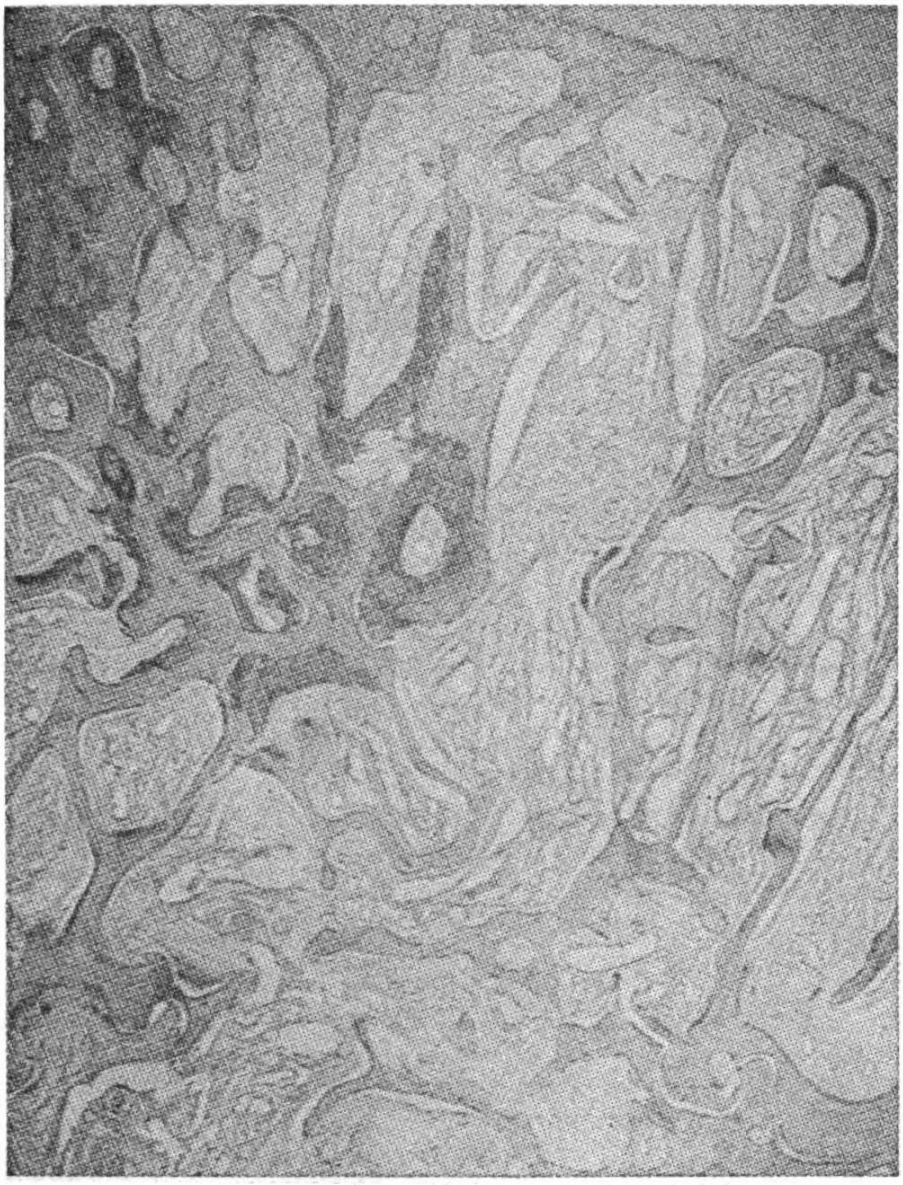

FIGURE 7.—Photomicrograph of one of the areas of atypical osseous tissue seen in the histological sections. The atypical osseous tissue is seen at the top of the photograph. The acellular fibrous tissue is seen throughout the trabecular spaces. (Reproduced courtesy of Journal of Bone and Joint Surgery.)

FIGURE 8.—Higher magnification of one of the areas seen in figure 4. Bone is seen in the lower left corner, with darker staining atypical osseous tissue adjacent to it. Fibrous connective tissue is seen in the remainder of the field. (Reproduced courtesy of Journal of Bone and Joint Surgery.)

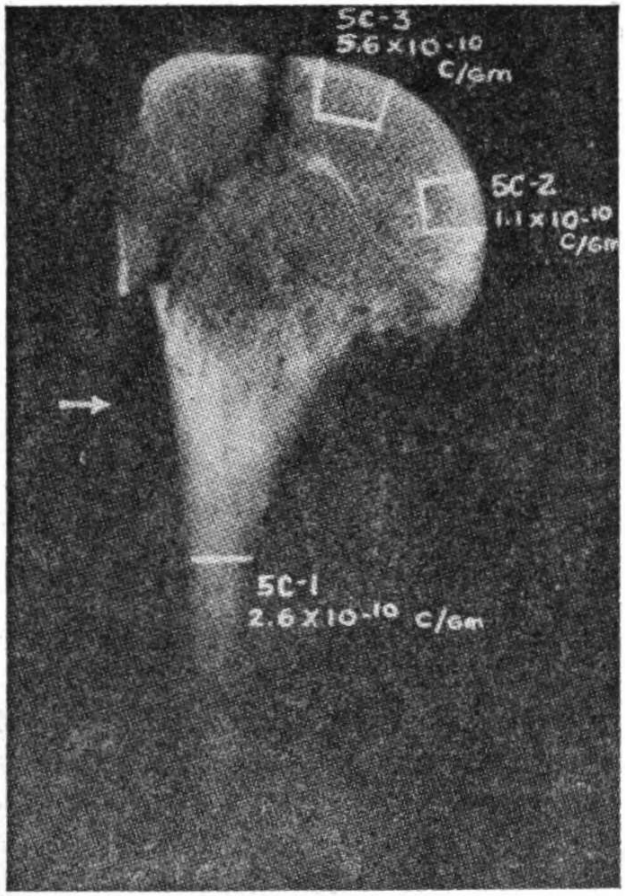

FIGURE 9.—Roentgenogram of a 2-millimeter section taken from the head of the right humerus. (See fig. 6.) Sections of bone, 5C–2 and 5C–3 were removed following the making of gross autoradiographs of this bone section. As will be noted in the figure, 5C–2 was taken from an area which seemed to have a large concentration of radium as shown by gross autoradiography; 5C–3 was taken from an area which seemed to have a small amount of radium as shown by gross autoradiography and a normal roentgenographic pattern. It should be noted that the radium content in RC–3 was five times the content in RC–2. Radium values are expressed as curies per gram of ashed bone. (Reproduced courtesy of the Journal of Bone and Joint Surgery.)

FIGURE 10.—Roentgenogram of the head of the femur of patient (R-34). The mottled appearance is the result of the atypical osseous tissue and hypertrophy of the trabeculae and is characteristic of the changes seen in cancellous bone following the deposition of radioactive elements (Review figs. 6, 7, 8, and 9). (Reproduced courtesy of Journal of Bone and Joint Surgery.)

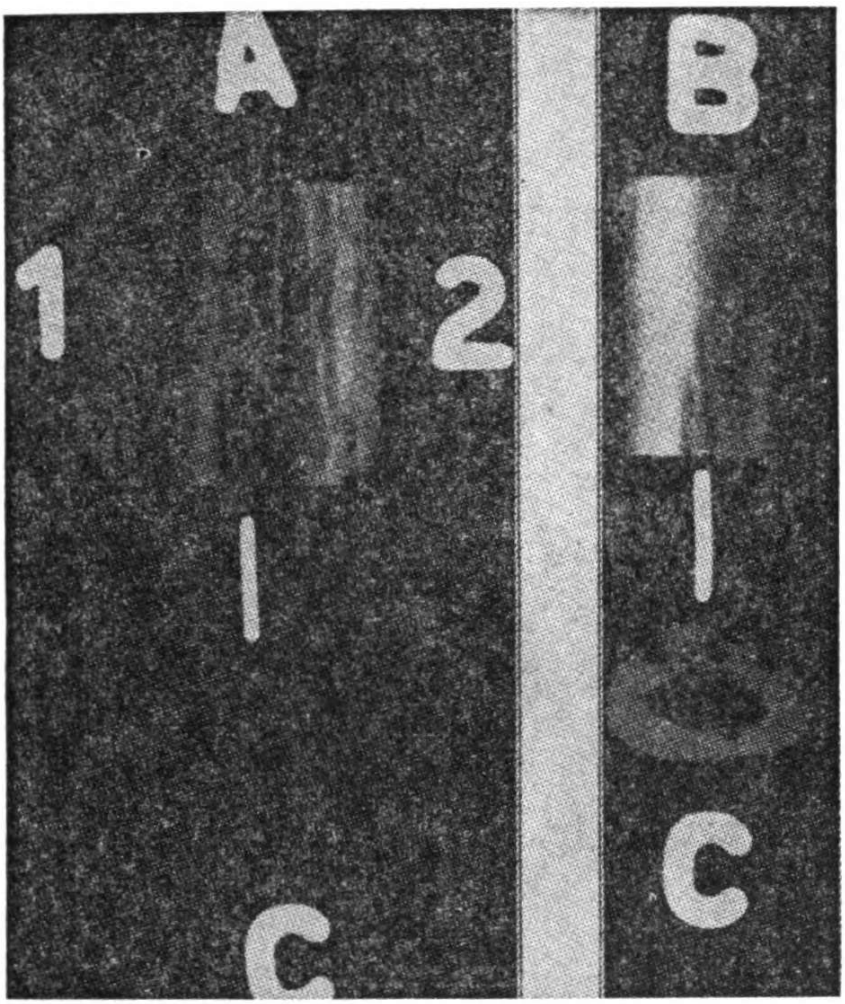

FIGURE 11.—Roentgenogram of a horizontal and vertical section of the fibula of patient (R–24). Note the "streaked" areas that are seen in the long bones as the result of well differentiated areas of destruction in the cortex. The pointers show the areas of destruction in the vertical and horizontal planes (Argonne National Laboratory Report, ANL 4666).

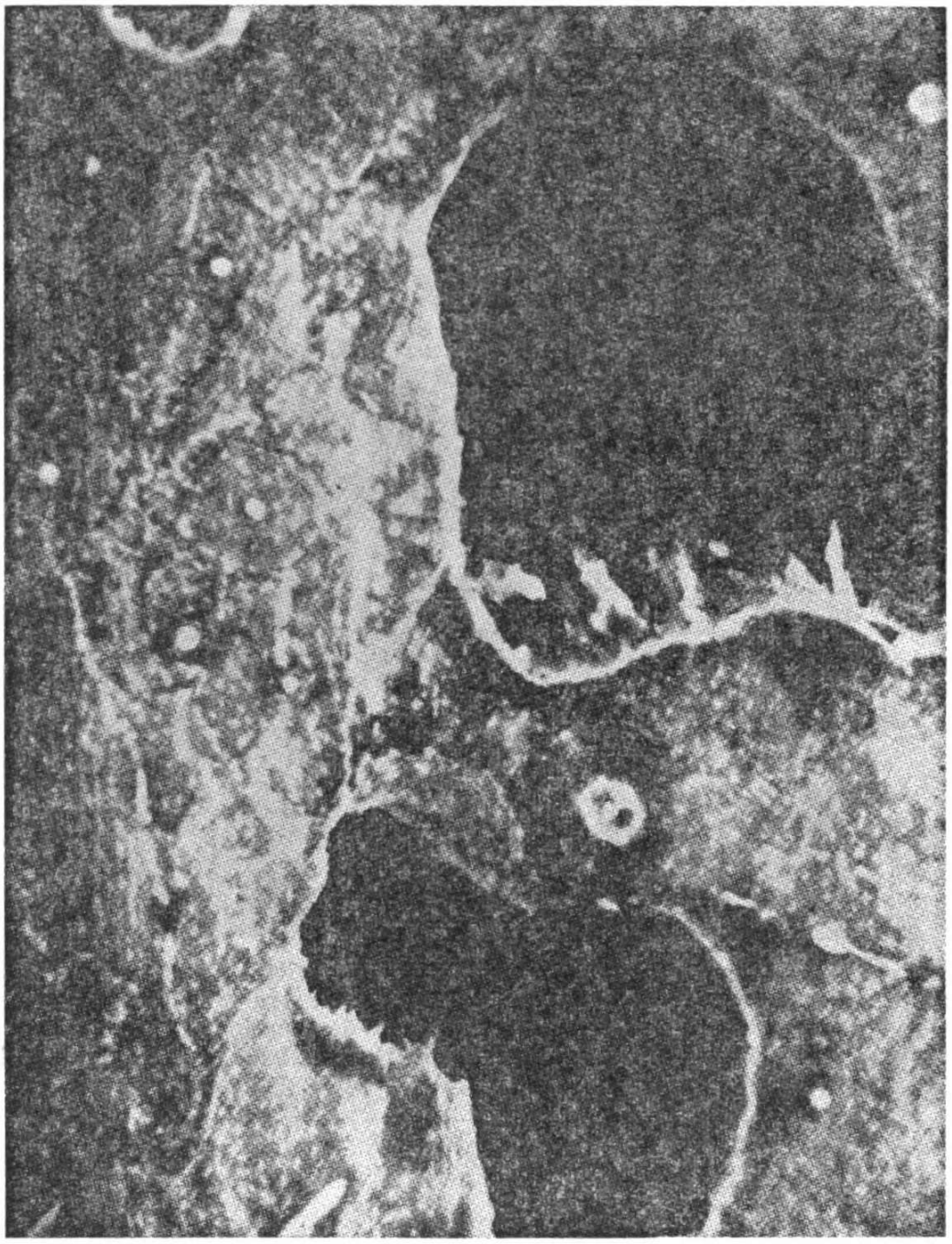

FIGURE 12.—Photograph of the areas of destruction seen in figure 11B. Note that the areas of destruction which give a "streaked" appearance to the long bones are about 3 to 6 times the diameter of the Haversian systems. The central canal in between the two macroscopic areas of destruction is about one-third the diameter of the Haversian system, while in the upper right corner there is a central canal that is enlarged to about two-thirds the diameter of the Haversian system (Argonne National Laboratory Report, ANL 4666).

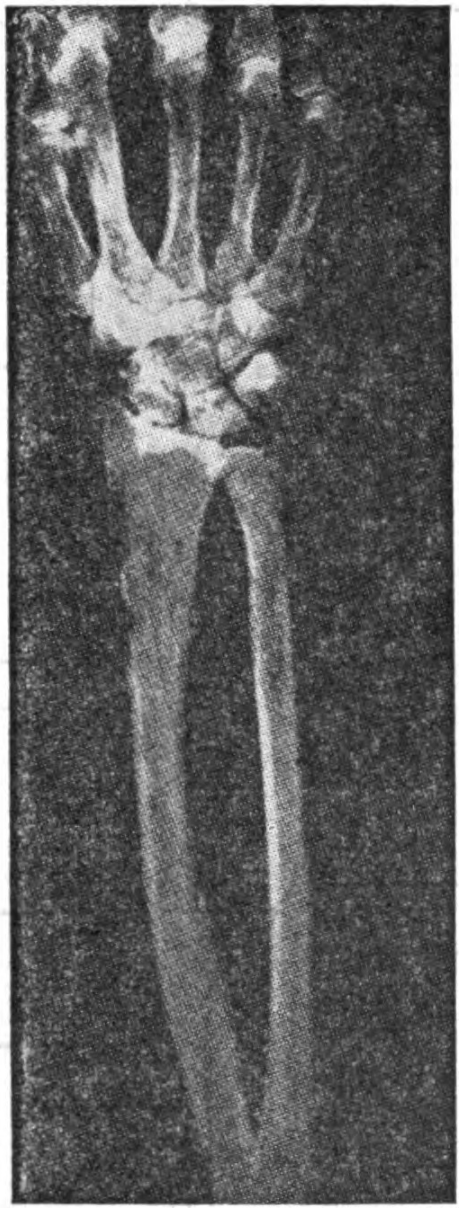

FIGURE 13.—Roentgenogram of the lower arm and hand of patient M. L. (L–27). The small areas of decreased density in the radius and ulna and bones of the hand give a streaked appearance. These small well differentiated areas of decreased density are characteristic of the deposition of radioactive elements. (Reproduced courtesy of American Journal of Roentgenology, Radium Therapy and Nuclear Medicine, 72: 842, 1954.)

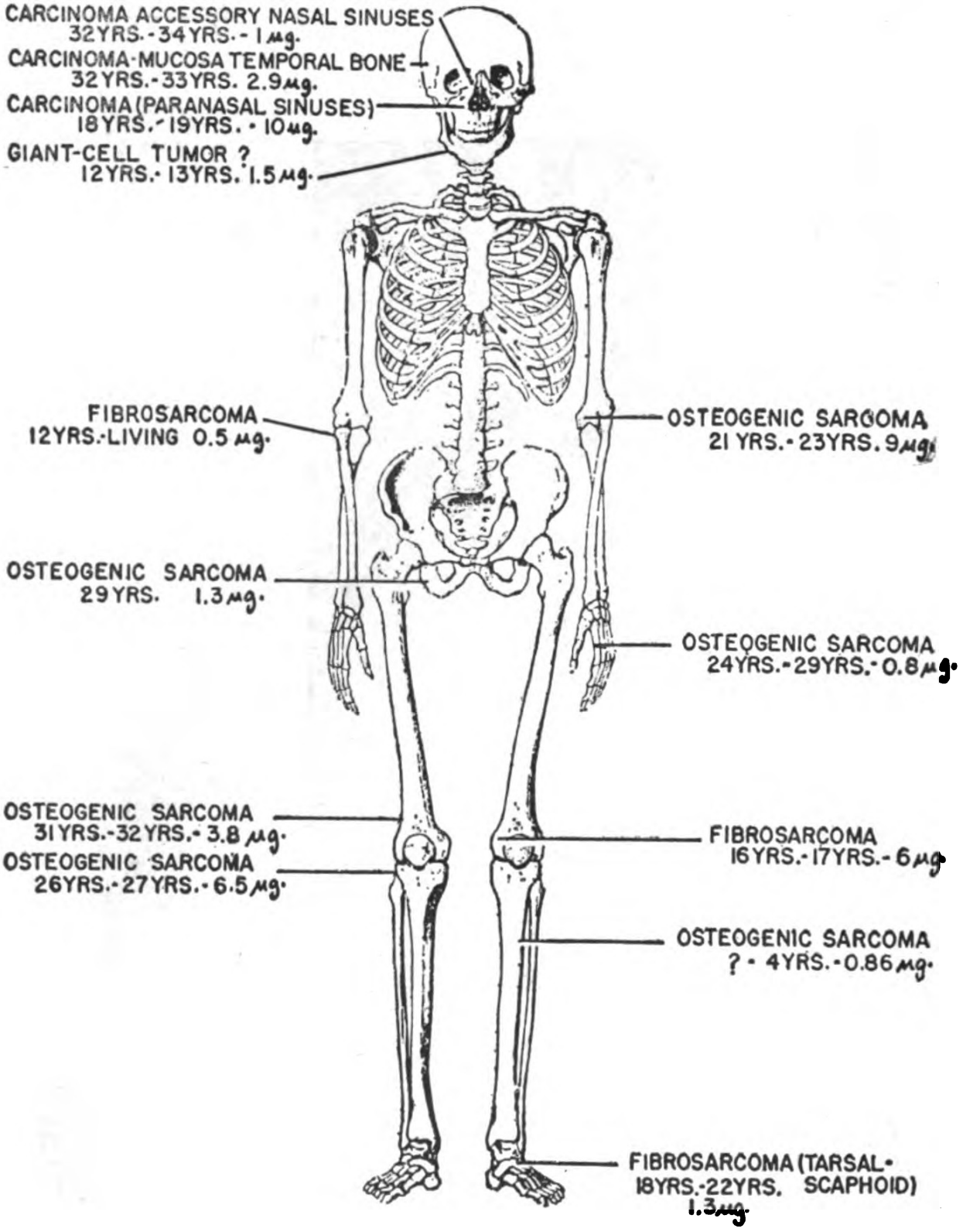

FIGURE 14.—Tumor formation in patients in Boston and Chicago investigations (1951). The five tumors which developed in the 50 patients who had received radium are shown on the right side of the skeleton and the 8 tumors which developed in 8 luminous-dial painters are shown on the left side of the skeleton. Two more tumors have developed in the patients who had received radium medically. These are not included. The type of tumor, the time from deposition to occurrence of symptoms, the time from deposition to death, and the amount of radium are given in each case. Reading from top to bottom, the numbers of the luminous-dial workers are 15, 20, 25, 16, 8, 14, 21, and 23; the numbers of the radium patients are 45, 17, 36, 18, and 24. (Reproduced courtesy of Journal of Bone and Joint Surgery.)

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ACKNOWLEDGMENTS

The author wishes to express his appreciation to Dr. William Rogers, editor of the Journal of Bone and Joint Surgery, Dr. Lawrence Reynolds, editor of the American Journal of Roentgenology, Radium Therapy and Nuclear Medicine, and to Dr. Paul Cannon, editor of the American Medical Association Archives of Pathology for granting permission to incorporate results of investigations published in their respective journals in this report.

From 1950 to 1952, the author was a postdoctorate Atomic Energy Commission fellow in the medical sciences of the National Research Council at the Argonne National Laboratory. He was assigned the responsibility for the clinical aspects of the investigation of individuals who had been given radium salts and people who had been employed as luminous-dial workers (1915 to 1930) and the responsibility for the correlation of the clinical aspects with the biophysical aspects of the investigation. This investigation was conducted by the following: Dr. A. M. Brues, Dr. L. D. Marinelli, Dr. W. P. Norris, Dr. R. J. Hasterlik, and Dr. A. H. Stehney, their associates and the author. The histopathological studies were made at the department of orthopedic surgery, University of Chicago, with Dr. C. Howard Hatcher. Detailed reports of the various phases of the investigation will be published.

The author was afforded the privilege of reviewing the clinical data of a similar investigation carried out at the Harvard Medical School and the Massachusetts Institute of Technology by Dr. J. C. Aub, Dr. R. D. Evans, Dr. L. H. Hempelmann, and Dr. M. S. Martland.

The Atomic Energy Commission fellowship, 1950–52, was under Dr. A. M. Brues, Director, Division of Biology and Medicine, Argonne National Laboratory.

Dr. L. D. Marinelli, Dr. W. P. Norris, Dr. A. H. Stehney, and their associates were responsible for the physical and radiochemical aspects of the investigation at the Argonne National Laboratory.

Dr. R. J. Hasterlik and Miss Ellen Shermont gave their assistance in the clinical and hematological aspects of the investigation at the Argonne National Laboratory.

Dr. Joseph Aub, Dr. Robley Evans, Dr. Louis Hempelmann, and Dr. Lawrence Robbins gave their assistance in the review of the clinical data of the investigation carried out at the Harvard Medical School at the Massachusetts Institute of Technology.

The late Dr. Dallas Phemister and Dr. C. Howard Hatcher of the University of Chicago gave their assistance in the orthopedic and histopathological aspects of the investigation.

Dr. Granville Bennett of the University of Illinois, Dr. Lent Johnson of the Armed Forces Institute of Pathology, and Dr. J. A. Turner of the Naval Medical School gave their assistance in the histopathological aspects of the investigation.

Dr. William Bloom and Dr. Franklin C. McClean of the University of Chicago gave their assistance in the histopathological aspects of the investigation.

Dr. James Arnold and Miss Lois Woodruff gave their assistance in the autoradiographic aspects of the investigation.

Dr. Paul C. Hodges and Dr. Russell Nichols of the University of Chicago and Dr. Robert Potter of Northwestern University gave their assistance in the radiological aspects of the investigation.

Dr. Edward Jerome of the Naval Medical Research Institute, and Mr. Sylvanus Tyler and Miss Joan Guerin of the Argonne National Laboratory gave their assistance in the statistical aspects of these investigations.

Dr. V. E. Archer of the National Institutes of Health gave his assistance in the preparation of the data on the elimination of radium.

Mr. Atlee Tracy of the Argonne National Laboratory and Mr. J. T. Stringer of the Naval Medical School were responsible for the photographs.

Mr. Melvin Runkel of the Naval Medical School and Miss Frances Fee of the Argonne National Laboratory prepared the illustrations.

A Study of the Dynamics of Strontium and Calcium Metabolism and Radioelement Removal[1]

PRELIMINARY REPORT, MAY 31, 1957

W. B. Looney,[2] C. J. Maletskos,[3] M. J. Helmick,[4] J. Reardon,[5] J. Cohen,[6] W. Guild,[7] and F. I. Visalli[4]

The classic studies of Aub, Evans et al.[1a] in 1938 in radioelement removal demonstrated that the renal clearance for radium was less than 1 percent in 24 hours. This finding suggested the possibility that direct radioelement removal from the blood might prove to be an effective way of eliminating radio-

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[3] Research physicist, Massachusetts Institute of Technology.

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[6] Visiting fellow in physics, Massachusetts Institute of Technology.

[7] Acting director, cardio-renal service, Peter Bent Brigham Hospital, instructor in medicine, Harvard Medical School.

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elements. Both the artificial kidney[3] and ion-exchange resins[4] afford means for radioelement removal, as well as providing an opportunity for extension of the work by Hastings and Huggins[4] on the mobilization of calcium in the circulating body fluids.

Preliminary in vitro experiments were performed to determine some of the parameters necessary to evaluate the feasibility of using either approach.

The first experiments were carried out on a simulated artificial kidney. Stable calcium, calcium 45, and ethylenediamine-tetraacetic acid were readily dialyzable under conditions similar to those in the artificial kidney (calcium being omitted for the purpose of the present experiments).[5]

In later experiments solutions of strontium 85 and calcium 45 were passed directly through ion-exchange columns to determine the effectiveness of a synthetic cation exchange resin in removing radioelements.

A total of 20 dogs has been studied, each dog having been connected to either the artificial kidney or ion-exchange column following the administration of strontium 85, strontium 89, and calcium 45. The ion-exchange column has been used in preference to the artificial kidney because of its more effective removal of calcium, its simplicity and potential adoption for more extensive utilization.

The ion-exchange resin offers a wide range of potential application to biological investigation. Appropriate adjustment of the cation concentrations in the column permits preferential removal of a particular cation of interest. The ability to place electrolytes of a biological system out of equilibrium affords an excellent tool for the study of the dynamics of both the stable and the radioactive electrolytes of the biological system.

The efficiency of radioelement removal as a function of time after administration has been determined by both single- and multiple-isotope methods. Between 30–40 percent of the radio-isotopes injected intravenously 1 hour prior to connecting the dog to the resin column can be removed during a 4- to 6-hour period. At 12 hours after injection, total removal of the radioelements decreased to about 6 to 12 percent, and at 24 hours to about 3 to 6 percent. After 3 days about 2 percent is removed. When the experiment was repeated in 2 dogs 1 week later, less than 1 percent was removed.

Analysis of the removal of the isotopes in three dogs indicated the following: About 80 to 90 percent of the dose is in a hypothetical compartment of bone which has a half time of removal of 8 to 16 hours; the remainder of the dose is in a compartment approximating in size the extracellular space and the half time of removal is 15 to 30 minutes.

To test the influence of serum calcium concentration on isotope removal, calcium 40 was infused intravenously at the same time as the resin perfusion. No significant decrease in radiostrontium removal has been found in 2 dogs in which the serium calcium levels of 6 to 8 milligrams percent were maintained. This result would be consistent with the hypothesis that the principal mechanism of radioelement removal by the ion-exchange column is cation exchange rather than enhanced physiological response from depressed calcium levels.

These studies demonstrate that the ion-exchange column and the artificial kidney are practical means for studying the dynamics of stable and radioactive electrolytes. No major contraindications have been found to prevent its adoption for clinical use.

Representative COLE. Mr. Chairman, if you do not mind, I would like to ask again, Dr. Looney, if you will interpret your expression that the assumption that the incidence of the effect of strontium 90 is proportionate to the magnitude of the dose. You have indicated that experience and studies led you to the conclusion that this is an overcautious—did you say overcautious?

Dr. LOONEY. Yes.

---

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[4] Hastings, A. B.: Studies on the Effect of Alteration in the Concentration of Calcium Circulating Fluids on the Mobilization of Calcium. Metabolic Interrelations, Transactions of the Third Conference, 1951, pp. 38–50.

[5] Looney, W. B., Maletskos, C. J., and Helmick, M. J.: Removal of Radiocalcium in Dogs, Progress Report, Radioactivity Center, M. I. T., Department of Physics, May 1956.

Representative COLE. Assumption?

Dr. LOONEY. Yes.

Representative COLE. What do you mean by that?

Dr. LOONEY. I mean that the present clinical information that we have would not substantiate the concept of a linear relationship, neither does it disprove it. If I had to weigh and balance these two factors, I would say that most of the information indicates that a certain amount of irradiation is necessary to produce bone tumors in man. However, this is based on the available information. It certainly is to be again emphasized that these tumors may be produced in man at a lower dose, and we have not been able to detect this at present. There are some methods of obtaining more information from clinical studies. I think they will be done.

To say this might be an overcautious assumption is probably not a good term, but I hope I have given you the reason for making the statement.

Representative COLE. Since your clinical evidence indicates that the incidence may be less in proportion to the magnitude of the dose, would that indicate the possibility of a threshold for the effects of strontium 90?

Dr. LOONEY. I would say that the present clinical information in man would not substantiate either conclusion.

Representative COLE. Based on clinical evidence?

Dr. LOONEY. Based on the evidence in man; yes, sir.

Representative COLE. You are not able to determine yes or no with respect to threshold?

Dr. LOONEY. No, sir; I am not.

Representative HOLIFIELD. Dr. Looney, how were you able to determine the amount of exposure these people had, in view of the fact it was years later before you were aware of their illnesses?

Dr. LOONEY. The patients in Chicago were found by reviewing the records of a mental hospital in which the patients were given radium. The files of the United States Radium Corp. were also made available to the Argonne National Laboratory, and we were able to get names and to locate these people by following names.

Representative HOLIFIELD. This did not obtain to those employed as radium painters?

Dr. LOONEY. Yes; it did.

Representative HOLIFIELD. Were you able to measure the dose they received at the time they received it by the residual amount in their bones when it was called to your attention?

Dr. LOONEY. The physical estimates were made by the physicists at Massachusetts Institute of Technology, and at Argonne National Laboratory. That is a physical area, and I would prefer to leave it to the physicists.

Representative HOLIFIELD. I was just interested to find out if we had an accurate estimate of the original dose. I know in the case of the Hiroshima and Nagasaki people that is one of the missing elements in our evaluation of the dose, that we do not know exactly how much they have received.

Dr. LOONEY. Yes. In regard to the patients in the mental hospital, we do have a record of the amount given, and the estimates at 6 and 12 months. Estimates were also made after 20 years. This is the

best available evidence we have in man. Based on this information we can make estimates of the original dose of radium in other people in which we find the radium 20 to 30 years after administration. The physical data on the luminous dial workers is confused by the fact that mesothorium and radiothorium were present in the paint. This presents a very difficult problem in trying to establish reliably the relationship of the clinical changes to the physical estimates of the radiation dose.

Representative HOLIFIELD. Thank you.

Representative VAN ZANDT. Dr. Looney, is it possible to leach out selectively the poison that has gotten into the skeleton of the body?

Dr. LOONEY. You are talking about removal of these radio elements once they are deposited in bone?

Representative VAN ZANDT. Yes.

Dr. LOONEY. We have been working on that in Boston in the past 2 years, trying to remove strontium 85, strontium 89, and radio calcium from bone. We found in the first hour we can remove approximately 30 or 40 percent of the strontium. This efficiency of removal rapidly declined, until after 2 or 3 days, it was less than 1 percent.

Other methods using chemicals to remove bone-seeking radio element have been attempted. There has been some progress in this field, but once the radio element is deposited in the bone, the chances are very remote that we will be able to remove it.

Representative VAN ZANDT. Thank you.

Chairman DURHAM. Doctor, figure 14, where you have the skeleton——(See p. 1188.)

Dr. LOONEY. Yes, sir.

Chairman DURHAM. Was any conclusion made as to why the carcinoma was more prevalent in the head and nose and mouth than other parts of the body?

Dr. LOONEY. You will notice that these people who developed these tumors were luminous dial workers. There has been the hypothesis that because the material was ingested and inhaled, rather than injected intravenously or given orally, that it might be the result of the local effect. But, I might point out that one of the radium patients has also developed a similar type of tumor. I have no readily available conclusion as to why this developed.

Senator HICKENLOOPER. Mr. Chairman.

Representative HOLIFIELD. Senator Hickenlooper.

Senator HICKENLOOPER. Just from a visual examination of the chart, it would indicate that one might assume the tumors occurred in proximity to the point of contact or ingestion of this radium treatment, or the radium material. In other words, with the luminous dial patients, as I understand it, they ingested this as a result of either inhalation or putting the brushes in their mouths to wet them so they could point them up and paint the figures on the dials.

Dr. LOONEY. Yes, sir; that is correct.

Senator HICKENLOOPER. And the incidence seems to be much greater in the nasal and throat area in those. On this chart there is no incidence of a tumor above the elbow in the strictly radium patient as differentiated from the luminous dial patient.

Dr. LOONEY. Yes. However, you will note it is a different type of tumor in the radium patients. You will notice these tumors are carcinomas in the luminous dial workers, and you will notice that the

sacromas of bone have occurred both in the luminous dial workers and radium patients.

Senator HICKENLOOPER. I am speaking purely as a layman who knows nothing whatsoever about this thing. A layman might be led to the conclusion from the chart that radium treatments might have been given in other parts of the body, but in the luminous dial workers the repeated incidence is at the point of ingestion very frequently.

Dr. LOONEY. This is an interesting observation, sir. I know this has created considerable comment as to whether there is a casual relationship among the people associated with these investigations. I am trying to point out both arguments for and against this. It is a different type of tumor that has developed in his area, and most of the tumors have been sarcomas of the bone.

Chairman DURHAM. Did the same type rays produce the three different types of sarcomas and carcinoma? You have three different types. Did the same rays produce all three types?

Dr. LOONEY. I think that the present available evidence is that any radiation has a similar effect biologically. So that it would be a combination of these three effects.

As far as radium is concerned, the majority of the radiation comes from the alpha particles, probably 90 or 95 percent.

Chairman DURHAM. Then the conclusion would be that beta rays or gamma rays would produce all types of carcinomas?

Dr. LOONEY. In sufficient quantities we must be aware of the quantitative aspects of the effect of radiation. I think it was brought out previously that the very sensitivity of physical measurements may lead to overemphasizing the effects of minute amounts of radioactivity.

We have a range of thousands of times between the amount of strontium 90 that is present, and the amount of strontium 90 that we calculate to produce equivalent energy to cause tumors in man from the available information on radium.

Representative VAN ZANDT. Mr. Chairman?

Representative HOLIFIELD. Mr. Van Zandt.

Representative VAN ZANDT. Dr. Looney, you have been talking about bone tumors. What about the utilization of radiation to attack these bone tumors?

Dr. LOONEY. Well, sir, I am not a radiologist, and this is in the field of radiology. I am aware of this, but I think you would get a much more competent opinion on this from some radiologist who is actively engaged in treatment.

Representative VAN ZANDT. Would the same thing apply to arthritis?

Dr. LOONEY. Yes, sir.

Representative VAN ZANDT. One more question, Dr. Looney. You have had many years of experience now in studying the body that has absorbed radiation from radium. Have you developed any kind of a preventative to this type of radiation?

Dr. LOONEY. No, sir, we have not yet. So far as I know, there has been no effective means of treatment.

Representative VAN ZANDT. Have you developed any type of a program that would prepare the body to resist radiation stemming from radium?

(Whereupon, at 1:10 p. m., the committee recessed, to reconvene at 2 p. m., of the same day.)

AFTERNOON SESSION

Representative HOLIFIELD. The committee will come to order.

Our first witness this afternoon is Dr. Ralph Lapp, a well-known physicist and author of books on radiation and related subjects. Dr. Lapp, we are pleased to have you before us this afternoon, and you may proceed.

STATEMENT OF DR. RALPH E. LAPP, WASHINGTON, D. C.[1]

Dr. LAPP. Thank you. Mr. Holifield, I received your invitation to testify before this committee while I was in Japan. I cut short my trip in order to attend the hearings. May I say that I appreciate very much this opportunity to appear here. I would like to add that I am very gratified that your investigations to date have thrown so much light on the problem of radioactive fallout.

I regret that the rate of accumulation of data in my files is so great that I have trouble in digesting it. I hope that during the next month I will have the time to analyze properly the amount of data that has been presented and that I hope I may be able to give you a later statement. I have been out of Washington for 3 months, and I have had trouble keeping up with things. Nonetheless, I am delighted to be here and testify before your committee.

Representative HOLIFIELD. We have permitted other witnesses to present additional data to substantiate their statements, and that same permission will be granted to you.

Dr. LAPP. I believe that these hearings will stand as a landmark in the history of our knowledge about this relatively new phenomenon.

Now I would like to add a comment about Dr. Libby. Appearing as I do after Dr. Libby, I would like to comment on his contributions to fallout. Dr. Libby has not only stimulated extensive research in fallout investigations such as Project Sunshine, but he has also taken the initiative in publication of his findings. I feel very strongly that he deserves a great deal of credit for his work on fallout. Were it not for Dr. Libby, we might well be confronted with a considerably smaller body of knowledge about fallout than we have today.

Mr. Chairman, I do not intend to read this entire document because it is too long, and I will skip over certain sections, with your permission, I might just mention that my interest in fallout extends over quite a period of years. I do not claim to be a great prophet with regard to the ultimate effects of radioactive fallout, since I think any examination of my publications about the Bikini shot of 1946 will

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[1] Nuclear Science Service, Washington 7, D. C. Physics, Buffalo, N. Y., August 24, 1917. Strong Foundation fellow, Chicago, bachelor of science, 1940, doctor of philosophy (physics), 1946. Instructor, Chicago, 1940–41; research associate metallurgical laboratory, Manhattan project, 1943–44; division director, 1944; assistant to laboratory director, 1945; assistant laboratory director, 1946; Argonne National Laboratory, 1946; science adviser, General Staff Research and Development Division, U. S. War Department, 1946–47; Deputy Executive Director, Atomic Energy Commission, Joint Research and Development Board, 1947–48; Executive Director, 1948–49; head, Nuclear Physics Branch, Office, National Research, Department of the Navy, 1949–52; Director, Nuclear Science Service, 1953–; industrial consultant and lecturer, 1953–; assistant, Chicago, 1940–41. A. A., physical sociologist, cosmic radiation; meson; bursts; showers; mass spectroscopy; radiological safety; nuclear radiation physics. (From American Men of Science, 1955.)

show that I was not terribly impressed with the phenomena of radioactive fallout at that time. I had to learn the hard way.

I might say that my interest in radioactive fallout was really stimulated by the 1954 Bravo test at Bikini. This was the test which resulted in radioactive contamination of the *Lucky Dragon No. 5*, a Japanese tuna trawler.

Based upon results which were made available largely by the Japanese, I began the publication of a series of articles on fallout at the invitation of the editor of the Bulletin of Atomic Scientists, and with your permission I will put these into the record, if you so desire.

Representative HOLIFIELD. They will be received and filed with the committee.

(The series referred to follows:)

November 1954, Civil Defense Faces New Peril
February 1955, Radioactive Fallout
June 1955, Radioactive Fallout III
November 1955, Global Fallout
September 1956, The "Humanitarian" H-Bomb
October 1956, Strontium Limits in Peace and War

Dr. LAPP. I am dividing my testimony into four parts: general remarks, local fallout, remote fallout, and I am so bold as to add some constructive proposals.

Under "general remarks," I would say I believe public confusion about fallout will continue to increase unless scientists can provide a quantitative or semiquantitative evaluation of the various hazards associated with fallout. Precision is probably not possible due to the nature of the hazards, and we may have to be content with numbers which vary by a factor of 2, 3, or even 10. This committee, I believe, has already performed a valuable service in narrowing the range of estimates made by individual witnesses.

Regarding disagreement among the scientists, I think that the public may well conclude that if scientists cannot agree upon hazard, then all is confusion. It would be nice if the scientists could all agree upon a quantitative estimate of the hazard, which could then be given out to the public. I maintain two unusual circumstances have combined to produce the current confusion on radioactive fallout.

First, the urgency of our times has focused attention upon problems for which science did not have textbook answers. Available knowledge was inadequate and research such as Project Sunshine had to be initiated to provide answers.

Second, the ordinary process by which scientists argue out their answers was interdicted by the complexity of the problem and also by secrecy. Scientists outside the Atomic Energy Commission have full-time jobs, as do those inside, and could scarcely be expected to tunnel into the complexities of the problem in a few leisure hours.

With regard to the responsibility of the Atomic Energy Commission, I think that if we consider these factors, the Atomic Energy Commission has responsibility for providing the outside world with the facts about fallout as promptly as they become available.

Representative COLE. Mr. Chairman, I wonder if Dr. Lapp would mind interruption while his statement proceeds?

Dr. LAPP. I would welcome interruption.

Representative COLE. We all agree with your statement that the Commission should make revelation of its information about fallout as promptly as it becomes available. Do you have any feeling that the Commission has failed in that responsibility, except insofar as fallout material may have a direct hearing on weapons information?

Dr. LAPP. Mr. Cole, I believe that your qualification there makes it difficult for me to answer the question. When you say "except insofar as it may have bearing upon weapons information," you, of course, bring into play the difficulty. May I answer it as specifically as I can with regard to the question of radioactive contamination of large areas.

I believe that the Atomic Energy Commission could have made available to the public far earlier than it did its report of February 15, 1955.

This is the report on the effects of high yield nuclear explosions. It took almost a year from the time that explosion occurred until this, I would call it nonquantitative report, appeared. It has taken additional time until the weapons-effects handbook—I believe called The Effects of Nuclear Weapons—will appear, presumably this month.

It is my personal opinion, Mr. Cole, that could have been done more expeditiously.

Representative COLE. But if it had been published earlier than 10 months after the test, would you not agree that any report would have been based on conjecture rather than on facts?

Dr. LAPP. No, Mr. Cole, I think the facts about this particular incident were fairly well known as of May 1954, and I believe if one looks at the record of the investigation of this committee—I am referring to the investigation of March 24, 1955, called The Atomic Energy Commission-Federal Civil Defense Administration Relationship—in this report published by your committee, the Atomic Energy Commission has stated that it had a summary report of the radioactive effects of the Castle series of tests available, I believe, in May of 1954. So based upon that testimony by the Atomic Energy Commission, I would say that they had the facts.

Representative COLE. The Commission, as you know—either the President or the Chairman of the Commission—did shortly after that test give a generalized report on the results of the test.

Dr. LAPP. Yes. It occurred, I believe, in my memory serves me correctly, on March 29 or 30, 1954.

Representative COLE. Some time in March.

Dr. LAPP. Yes. I personally do not feel that report was an adequate or quantitative evaluation of the hazard.

Representative COLE. Of course, it was not. It was not intended to be. But it was a general report to the public as quickly as general facts could be made known.

Dr. LAPP. It was a general report.

Representative COLE. As to the detailed lessons based on facts as they were observed might necessarily involve a period of time. It would seem that a period of 11 months, considering the importance of the subject under study, was not inordinate.

Dr. LAPP. I think we may disagree how long would have been required for this. I might say, Mr. Cole, if I am permitted a moment to look into my own notes here—that so far as I personally am concerned,

based upon data made available from the Japanese, I was able to publish in November 1954 in what I would call semiquantitative form an analysis of the hazard, and again in 1955 I was able to publish in the Bulletin of Atomic Scientists in February—I am sure I have it here, but it just escapes me—let me quote from memory.

Senator ANDERSON. I think I have a full file of the bulletins.

Dr. LAPP. It is the February 1955 issue.

Senator ANDERSON. That is probably the one I do not have.

Dr. LAPP. In this issue I believe I presented a quantitative estimate of the hazard which was more detailed in numbers than was the Atomic Energy Commission's release of February 1955. The situation, I realize, is difficult if one wishes to make a precise statement. But I do not think it was a precise statement that was needed. I believe something which would indicate the general magnitude of the hazard which civil defense faced would have been important.

By the way, I will come to this point in connection with later testimony.

May I continue?

Representative HOLIFIELD. You may proceed.

Dr. LAPP. I believe, then, that scientists, technicians, and officials of the Atomic Energy Commission must present reasoned and careful estimates of the hazards based upon factual knowledge. Reckless or nonsubstantiated statements do a disservice to the Atomic Energy Commission and to the Nation.

Representative VAN ZANDT. Dr. Lapp, you say reckless or nonsubstantiated statements do a disservice to the AEC and to the Nation. You make this statement under the title "The Responsibility of the Atomic Energy Commission." Are you charging that the AEC spokesmen made reckless or nonsubstantiated statements?

Dr. LAPP. I believe that the examples I have given here, particularly when we have a man like Dr. Richard Doan coming to Tokyo only last month stating that the bomb test did not have the slightest possible effect—I would be happy to place the entire quotation in the record—upon humans, that is nonsubstantiated. I believe it constitutes what may be regarded in the light of the great world importance of atomic energy as amounting to a reckless statement.

I have not charged that Dr. Libby made a reckless statement here when he said, "However, as far as immediate or somatic damage to health is concerned, the fallout dosage rate as of January 1 of this year in the United States could be increased 15,000 times without hazard."

Representative VAN ZANDT. Dr. Lapp, do you have all available information in this field?

Dr. LAPP. I have such information as is available to the public.

Representative VAN ZANDT. But you do not have the reservoir of information available that Dr. Libby has, do you?

Dr. LAPP. If I am to believe from Dr. Libby's testimony this morning that it was all unclassified, I would say that I must have.

Senator ANDERSON. It is either classified or you have it?

Dr. LAPP. Yes. I am sorry you are asking me something that is difficult for me because I simply do not know whether or not there exist things which are buried in secrecy.

Representative VAN ZANDT. I am not worried about classified information. I regard Dr. Libby as a member of the Atomic Energy Commission and therefore having at his fingertips all possible informa-

tion with access to AEC files. Therefore, when he makes a statement he must certainly make it based on the most comprehensive information available.

Now, I am asking this question: Do you have comparable information?

Dr. LAPP. No, sir.

Representative VAN ZANDT. By making the critical statement regarding the AEC spokesmen, in effect you compare yourself with Dr. Libby from the standpoint of availability of information. Therefore, it amounts to this, does it not, that it is simply your own opinion?

Dr. LAPP. It is my opinion based upon the facts which Dr. Libby has presented.

Representative VAN ZANDT. That is all, Mr. Chairman.

Dr. LAPP. I might say I can only go on what Dr. Libby has stated plus my knowledge of science and physics.

Representative PRICE. Mr. Chairman, may I ask Dr. Lapp a question?

Representative HOLIFIELD. Yes.

Representative PRICE. Is it merely your personal opinion, or is it your opinion based not only on information made available to you, but on consultation and association with other scientists?

Dr. LAPP. I have consulted with many scientists. I may not say in the specific preparation of this document because I was working until the early hours this morning doing this. I have in the past number of years consulted with many scientists of considerable note. I do not pretend to speak for scientists. But I might say this, Mr. Price; I have been encouraged by scientists.

Representative PRICE. You do not feel that you are alone in these opinions?

Dr. LAPP. I am alone right now, sir.

Representative PRICE. I do not think you have been alone. I think others have testified pretty close to the testimony that you started out to give here. I do not think you are alone.

Dr. LAPP. I would like to make this statement, Mr. Price. I have come here today to try to present such observations as I can to be of help to this committee. I do not want to be put in the position of challenging every statement that Dr. Libby makes.

Representative PRICE. I did not assume that you were challenging everything. The point I wanted to draw out was whether or not these were just strong personal convictions on your part, or whether or not you had arrived at your conclusions not only through the examination of the material furnished to you but through your personal studies and association with other leading scientists not only in this country, but in other parts of the world.

Dr. LAPP. I have recently consulted with scientists in Japan. I might say that one of the useful things that I accomplished in discussing these problems with the Japanese was that I think I explained some of the strontium measurements to the Japanese and tried to reconcile Dr. Libby's data with their own. I think it was useful because many of their figures were quite high as compared with Dr. Libby's. Although I am not yet sure that they believed everything I have said, I have been in correspondence with them since and I have tried to bring about some agreement in this field.

Again, I state that I personally owe a great debt of gratitude to Dr. Libby as I think the country does in providing us with these very valuable data.

Representative PRICE. I think we all agree with you on that. I think you were interrupted before you were permitted to give the examples that you were going to give on statements you referred to as perhaps "reckless" statements.

Dr. LAPP. I think we must interpret this and perhaps qualify it somewhat. That is, we are dealing now with statements made to the public. This is an area in which I have had some experience, namely, in the translation from the world of megacurie and the megaton into the world of ordinary understanding. I think Congressman and Senators know how difficult this translation can be. Dr. Libby's speeches have not always been masterpieces of simplicity for the press to understand. I have in many cases attempted to translate these speeches so that they might be more useful.

Senator ANDERSON. Doctor, now that you have had a chance to refresh your memory as to your fallout data, do you find you reported in the February 1955 issue of the Bulletin of the Atomic Scientists?

Dr. LAPP. Thank you. It was in the February issue of 1955 that I presented a quantitative estimate of the radiation exposure. I believe this curve showing cumulative dosage from fallout agrees with the data which are now presented some 3 years later in The Effects of Nuclear Weapons. I am going to testify about this.

Representative HOLIFIELD. You made that chart without access to classified information?

Dr. LAPP. The basis of making this chart was, if I may be permitted—this is a complex technical matter—to give you a complete explanation.

Representative HOLIFIELD. Make it simple.

Dr. LAPP. I have here the reports of the Japanese scientists. These are called Research in Effects and Influences of Nuclear Bomb Test Explosions. I want to point out that this has been published only recently, but it contains many of the scientific papers of 1954 upon which I based my deductions.

These reports might just be illustrative to show how much other countries are publishing. These reports are the research in the effects and influences of nuclear bomb test explosions in two volumes compiled by the Committee on Compilation of the Report on Research and Effects of Radioactivity, published in Japan for the Society for the Promotion of Science. There are about 1,800 pages of data in these reports.

Representative HOLIFIELD. The chart that you gave in February 1955 has stood up, then, against challenge, has it?

Dr. LAPP. I believe so. I have discussed this with Dr. Eugene Wigner, who is a scientist of outstanding note. We were much concerned about the tail of the curve. This tail of the curve is quite important because it is upon the tail of the curve that the long term persistence of radioactivity will depend. At the time I talked with him, Dr. Wigner had his doubts about how this tail may be extrapolated.

My own feeling is the recent data of Dr. Dunning, contributed to this committee, support this curve.

Representative COLE. Dr. Lapp, you will concede that your report of January 1954—or is it February?

Dr. LAPP. I think it came out in February.

Representative COLE. That was the same month that the Commission's report came out.

Dr. LAPP. Yes, Mr. Cole.

Representative COLE. But your report was based on studies of exposure to the Japanese fishermen.

Dr. LAPP. Yes.

Representative COLE. Whereas, that was only a part of the persons who were exposed to radioactivity. Is that not correct?

Dr. LAPP. That is correct, Mr. Cole.

Representative COLE. There were other people, the Marshallese, who were also exposed.

Dr. LAPP. That is true, but the data which I am concerned with is not data on people, but rather the actual number of curies of radioactivity deposited on the surface of the Lucky Dragon.

I might say, by the way——

Senator ANDERSON. Your report came out in the February issue which reaches the subscribers ahead of the first of the month. The Commission report came out in March and followed by a considerable period of time the publication of the data that you have there, did it not?

Dr. LAPP. I believe there was a difference in time of about 8 days. I prepared this, by the way, in December of 1954. I again received a tremendous amount of help from Dr. Libby's first speech of December 1954. It was very useful to me. I really feel that Dr. Libby has contributed immensely to this whole phenomenon of fallout.

Representative COLE. Mr. Chairman, before Dr. Lapp leaves this question of the responsibility of the Commission to make factual publication of its information, Dr. Lapp has indicated three examples which in his opinion represent a failure to fulfill that responsibility of frank and honest statements. He has characterized them as reckless and nonsubstantiated by the facts.

Dr. LAPP. Not all the statements.

Representative COLE. I notice you made an exception with respect to Dr. Libby's statement that it was not reckless, which leaves the conclusion that his statement nevertheless was unsubstantiated by the facts.

Dr. LAPP. With respect to that, Mr. Cole, I would wonder whether or not in the light of the testimony that has been received by this committee that Dr. Libby would still wish to subscribe to the statement that the fallout dosage rate as of January 1, 1955, in the United States could be increased 15,000 times without hazard.

Representative COLE. I am not seeking to argue with you. I am simply trying to establish clarity in the record.

Another question is this. These three examples which you have enumerated, do they constitute the only instances which have come to your attention where employees or representatives of the Commission have made statements on this subject which you feel were perhaps reckless to some degree, or not substantiated by the facts?

Dr. LAPP. No, Mr. Cole. I would have to go through my files. I believe there are many more.

Representative COLE. But would you agree that these are the most glaring instances of recklessness on the part of such people?

Dr. LAPP. Until I really combed my files and did a thorough job I can't answer the question.

Representative COLE. It would seem from the fact that these impressed themselves on your memory, and that you have cited them would indicate that they were the most extreme instances.

Dr. LAPP. No, I would not believe that. I think I cite them because they were readily at hand when I prepared this testimony.

Representative COLE. On this point, Mr. Chairman, it would seem to me that in fairness to these individuals whose responsibility has been questioned in this regard—Dr. Eisenbud and Dr. Doan—they should be given an opportunity to give whatever explanation they may have by way of defense against the charge of having issued a reckless and unsubstantiated statement.

Representative HOLIFIELD. I will respond to that. They will be given this opportunity if they care to.

Representative VAN ZANDT. Dr. Lapp, I am not doubting your ability as a physicist nor am I challenging your right of opinion, but for the first time during the course of these hearings we have the integrity of a Government agency challenged. That is what it amounts to. It has changed the complete tone of these hearings as far as I am concerned. We are not sitting here as an investigatory committee. We are sitting here for the purpose of trying to find the answer to this radiation problem. I think you would make a great contribution to these hearings if you would delete from your statement that charge you have made against the AEC and certain physicists employed by them.

Senator ANDERSON. I thought we were going to hear their replies to the charge. I think if Dr. Eisenbud will come in and prove as they laid down these criteria of proof the other day, that fallout to date from all tests would have to be multiplied by a million to produce visible deleterious effects, except in areas close to the explosion, that it would be very interesting.

Dr. Libby says he does not know what will happen when this comes out of the stratosphere. If Dr. Eisenbud would give us a short statement proving that it will have to be increased a million times, I think that would be very interesting. It would contradict a great deal of testimony from the Atomic Energy Commission itself.

Representative COLE. Mr. Chairman, I would point out by way of important emphasis that this statement of Dr. Eisenbud was made on March 20, 1955.

Dr. LAPP. Yes.

Representative COLE. So whatever response he may have to make by way of justification or explanation will have to be predicated as of that day, and not as of today.

Dr. LAPP. I would like perhaps to amplify this one bit, that is, that when I talk of the use of the word "reckless," there, I am speaking in terms of how the noneducated public will interpret statements made to them. That was my only purpose. I am not challenging the integrity of those people.

Representative COLE. Dr. Lapp, would you assert that the Commission's spokesmen in the scientific field were the only scientists who have made reckless and unsubstantiated statements on this problem?

Dr. LAPP. I would not. Reckless statements have been made by scientists who are not in the Atomic Energy Commission.

Representative COLE. Would you also say unsubstantiated statements were made by scientists who are not in the Atomic Energy Commission employ?

Dr. LAPP. I would readily agree to that.

Senator HICKENLOOPER. Mr. Chairman, may I ask Dr. Lapp, have you examined the full statements of Dr. Eisenbud and Dr. Libby and Dr. Richard Doan that you referred to here?

Dr. LAPP. Yes, sir.

Senator HICKENLOOPER. So it is based on that examination of the full statement that you quote out of context here?

Dr. LAPP. Yes, sir.

Senator HICKENLOOPER. And draw your conclusions that these add up to what you call reckless statements?

Dr. LAPP. Yes.

Senator HICKENLOOPER. I think perhaps one might argue that there is a degree of recklessness in dogmatic statements even by yourself in drawing these conclusions, not having been closely associated with the investigation of the data involved, and I rather question the advisability of the use of the word "reckless" in this rather difficult and quite ramified and uncertain field.

Senator ANDERSON. Dr. Lapp, would you not agree with me that there is a possibility that Dr. Eisenbud might not have been correctly quoted in the newspapers? I say that because Dr. Eisenbud gave us what I regarded as extremely fine testimony in his appearance before the committee. I thought it was scientifically based and carefully put together, and you have spoken very highly of Dr. Libby. I share your high regard for him, and I would hope that you might express to me how you feel about Dr. Eisenbud. I think he is a very capable and fine man. I hope that would sort of find some response in your system also.

Dr. LAPP. I was much impressed with the statement he made before this committee. The statement he made was in the Sunday News for New York of March 20, 1955. I believe that this illustrates one of the problems, Mr. Anderson, of the scientists and the press. He is really responsible for being careful in issuing statements to the press. I am not in any away attempting to attack the integrity of any people. I merely point out the impact which this will have upon the public.

So far as world interest in fallout is concerned—I will try to read this quickly—the committee may be interested in my observation that fallout has become an acute weapon of propaganda. For example, I found the Japanese scientists are actively studying the radioactivity of their tea, because of the assertion from the Chinese mainland that Japanese tea is radioactive. Apparently fallout does not occur in China. Some people in Japan are so keenly aware of fallout——

Representative HOLIFIELD. I think you should clarify that facetious remark, because in the print your manner of delivery might create the impression that you have said that it does not.

Dr. LAPP. I am sorry. It seems to me that here is an example of how radioactive fallout can be used as a weapon of propaganda in which a country which might stand to gain from its sale of a product accuses another country of having radioactive tea, and forces that other

country to engage in fairly laborious research project to find out how radioactive their tea is.

I visited this laboratory where tea was analyzed and saw the vast quantities of tea tested for radioactivity. I cite it merely as an example of how this can be used.

Representative COLE. What were the results of those tests?

Dr. LAPP. The tests are still underway. I believe the results will be given out within the year. I did not find any unusual radioactivity from the contact I had with Dr. Shiokawa. Some people in Japan are so keenly aware of the fallout that they actually take showers after being out in the rain.

There was a great public outcry against the British Christmas Island tests, but there was no great demonstration against the Soviet tests. It seems to me that this is a great victory for psychological warfare experts when they can induce selective sensitivity to fallout.

Representative COLE. Dr. Lapp, since you were there in Japan at the time of these tests or immediately prior to the tests, can you account for the fact that there was such a striking demonstration against the Christmas Island tests and yet there was no equal or even slightly proportional demonstration against the Russian tests?

Dr. LAPP. I might point out in all fairness that so far as the Japanese Government was concerned, they did protest the Soviet test, too, but as far as the public demonstration was concerned, I know of no such demonstration against the Soviet test. I think, however, one can give a partial explanation for this. This is in the fact that the Japanese people were immensely affected by the radioactive fallout in the fishing areas of the Pacific. After the accident of March 1, 1954, the Japanese Government went to considerable effort and expense to monitor the fish supply, which is a great source of protein for the Japanese. I think therefore they associated with the Christmas Island tests an effect upon their food supply.

Representative COLE. If that is so, how do you account for an equally demonstrative representation against the tests here in Nevada?

Dr. LAPP. I was not aware that they had such an outcry.

Representative COLE. You must have been aware that some Japanese people stormed the gates of the American Embassy in Tokyo in protest against the Nevada tests.

Dr. LAPP. I am sorry. I was traveling about Japan a good deal and perhaps I missed this one, Mr. Cole.

Representative COLE. This only occurred within the past month.

Dr. LAPP. I see. I did not know about this.

Representative COLE. Yes.

Dr. LAPP. I cite my reason as one factor.

Representative COLE. It is understandable that the Japanese people should be unusually sensitive to the hazard of radioactive fallout. That is very understandable. But it is difficult for me to understand why they should distinguish between the hazard of the British or American fallout without apparent protest to the hazard from Russian test fallout. Do you have any explanation for it?

Dr. LAPP. I could not profess to be extremely competent in this regard. I believe, however, there is a Communist Party at work in Japan. I believe that they use radioactive fallout as a political weapon. In fact, I believe that in the case of the survivors of the

Lucky Dragon, that their families were visited by representatives of the Communist Party who promised them money by way of being helpful. To my knowledge they never showed up with the money, but they promised them money.

Representative COLE. Were you not told when you were in Japan, as I was, that the newspaper reports of the Russian tests indicated to the Japanese people that the Japanese scientists had detected some unusual turbulance in the atmosphere? It was characterized as such, with no direct reporting that this was radioactive material. It was simply tagged as an atmospheric disturbance, whereas the press described the British and American tests as radioactive contamination of the air.

Dr. LAPP. I think they are probably allergic to American bomb fallout. This is rather interesting, in view of the fact that the data given to me by the Japanese show that some 70 percent of the total gross activity of the fission debris falling upon Japan is of Soviet origin, 20 percent from the Pacific tests and 10 percent from the Nevada tests.

Representative COLE. That was given to you by Japanese scientists?

Dr. LAPP. Yes.

Representative COLE. Have you ever seen that conclusion published in the Japanese newspapers?

Dr. LAPP. I gave a number of interviews when I was in Japan, and I pointed this out, but I unfortunately do not read Japanese, and do not know whether they reported it. It may have been. I stated the fact that there is greater fallout on Japan from Russian tests than from United States tests or United Kingdom. I am unaware that it was published. It may have been.

Representative COLE. This is a rather remote and roundabout route by which to provide these conclusions for the information of the Japanese people. I can hope that the Japanese reporters who are present here today may report to their respective newspapers published in Japan the conclusion which was given to you while in Japan, a conclusion by Japanese scientists—that their tests of the contamination of the atmosphere over Japan was caused 70 percent from the Russian tests and the balance from American and British tests.

Dr. LAPP. I think, Mr. Cole, I hope that these facts are reported in the Japanese press. I have reason to believe they will be. I think it is entirely reasonable that the fallout upon Japan should be predominantly from Russian tests because of the greater tropospheric fallout which Dr. Libby explained this morning. The greater tropospheric fallout from these tests will occur from the Russian tests. Because of the fact that they are in the air mass trajectory from the Soviet test region, this fallout will occur upon Japan sooner than upon other parts of the world. Because of the freshness of the fallout, there will be a greater radioactivity.

Representative COLE. That being a scientifically provable fact, then, is it not appropriate that the Japanese should be far greater concerned over Russian tests and the hazard of Russian tests than the tests by the United States and United Kingdom?

Dr. LAPP. If I were a Japanese I certainly would be.

Representative HOLIFIELD. It is not strange, as far as I know that the Atomic Energy Commission has never revealed this very important fact, and that the first knowledge I have of it is as of today? If

they have revealed it, I am unaware of it. We are being subjected to propaganda as you have stated by the Communist Party seizing on the part of this technical information which is to their advantage, and using it in Japan. Why is not this a piece of scientific fact that could be used on our part and let the world know that 70 percent of the fallout on Japan comes from Russian origin, if that is a true fact.

Dr. LAPP. Mr. Chairman, I am trying to strain my memory to remember this, but I believe that the National Academy of Sciences report contains data on relative fallout. I am trying to remember if it was on Japan. I think that it does contain data on this, but it is probably buried in the scientific literature.

Representative HOLIFIELD. I have read the report fully, and if it is in there, I have either forgotten it or I could not understand it, the way it was stated.

Dr. LAPP. I do not think it was in the summary or general report, but was in the greater compilation of the appendix, the physical measurements.

Representative HOLIFIELD. But a report of the American Academy of Sciences is not a publicized report of the Atomic Energy Commission.

Dr. LAPP. I might say this. I cannot remember any Atomic Energy Commissioner making a point of this in his speeches. I read most of the speeches quite carefully. I do not remember this being made.

Representative COLE. You do not know whether this information has been made available or known to the Commission, do you?

Dr. LAPP. I could not state positively.

Representative COLE. Of course, if the Commission had wanted to or happened to it could have found the same information which was given to you. I do not question that. But there is no evidence, or is there, that the Commission had this information?

Dr. LAPP. I am sorry, I am not competent to discuss that.

Representative COLE. Mr. Holifield referred to this conclusion of yours, or this information which was given to you as having an element of propaganda. I am sure he did not intend to use that word in the strictest sense, because this information in my opinion is not propaganda. This is a statement of the scientific facts as resulting from the examination of competent Japanese scientists.

Representative HOLIFIELD. I will accept the gentleman's amendment, if he will allow me to substitute the word "publicity" in the place of "propaganda."

Representative COLE. Yes. It should be publicized generally, and widespread, and that is why I am talking about it as much as I am in the hope that the reporters who are present will make certain that it is publicized fully and in all of the newspapers in Japan.

Representative HOLIFIELD. I had assumed that Project Sunshine was all over Japan, too, and we knew about this.

Representative VAN ZANDT. Mr. Chairman, I would like to ask Dr. Lapp another question.

Dr. Lapp, the Japanese scientific documents you have at your elbow, have you gone through them as yet?

Dr. LAPP. I have gone through a great many articles, yes. Some of them did not interest me too much, so I skipped over them. I have gone through a great many.

Representative VAN ZANDT. Dr. Lapp, do you think all the information contained in those Japanese documents is original or has somebody borrowed the information from the American scientific family?

Dr. LAPP. I would say that much of it is original and of course scientists borrow wherever they get the information. That is the nature of science. It is an international community. There are a great many references in each paper to the United States reports, and to other nations as well. I think that the United States reports predominate.

Representative VAN ZANDT. In other words, the volumes contain much United States data?

Dr. LAPP. No, they reference United States data. For example, if we have a radiochemical technique for detecting a particular radio element, then they would use the technique and reference this. I want to point out, however, that the Japanese scientists, despite the fact that they are not many in number as compared with other nations, have quite a history of excellence. I had the great pleasure of talking with Professor Kimura only two Sundays, ago, and he was very kind to show me some of his original work, and I know it was of very high quality.

Representative VAN ZANDT. Dr. Lapp, have you had made available to you a copy of a Russian scientific document entitled, "Preliminary Data on the Effects of Atomic Bomb Explosions on the Concentration of Artificial Radioactivity in the Lower Atmosphere and Soil"?

Dr. LAPP. I have not. (See p. 1209.)

Representative HOLIFIELD. It was just called to my attention by the chairman that one of our outstanding scientists in California is an American Japanese.

Dr. LAPP. The Japanese scientists are particularly competent in the field of theoretical physics. One of the greatest theoretical physicists in the world is Professor Yukawa, famous for his discovery of the meson.

It is inherent in the very nature of the biological research into the effects of radioactivity upon humans that a high degree of accuracy is not attainable, especially on human experience basis. As Dr. Langham, of the Los Alamos Laboratory, has testified, human experience with retention of radium 226 is the basis for setting upon a maximum permissible concentration for radiostrontium. Yet our actual experience is confined to a small sample of acutely exposed individuals and a small sample of less acutely exposed people.

Actually, our concern should focus not upon acute effects in man, which are highly unlikely from peacetime bomb testing, but rather with the chronic, debilitating, long-term effects from irradiation of humans. We must be conscious of the need to appraise long-delayed effects, say 50 years after entry of radio elements in the body. Here our knowledge is quite limited.

SECTION F. RADIATION LIMITS FOR A GLOBAL POPULATION

I would like to stress the fact that consideration of safe limits for irradiation of the world's population is essentially a new problem. Prior to the awareness of global fallout, the International Commission on Radiological Protection made its recommendations for those who would be exposed to radiation in pursuit of their occupation.

Such groups initially were numbered in the hundreds and then in the thousands as atomic energy came of age. Individuals within such groups were healthy adults exposed to known and restricted hazards; they were subject to administrative controls and medical supervision.

In setting up limits for a total population, we must take into account the varying radiosensitivity of individuals, the complete spectrum of age, the persistence of the hazards, the lack of medical control, the varying degrees of health of people, and the variety of their diet. Yet it was not until last year that the Atomic Energy Commission introduced the difference between an occupational MPC and a global MPC into its releases on fallout.

May I explain that briefly? Dr. Libby, in his speeches referred always to the MPC. I am not accusing Dr. Libby of deliberately trying to mislead anybody, but, from the standpoint of the ordinary layman reading these things, there was no distinction between a maximum permissible concentration for occupational workers and for the world population. I think this is one of the things that is necessary when you are putting information out to the public—that you must distinguish between these different units.

Representative COLE. Why is that, Doctor? What is the difference whether the individual absorbs or is exposed to the maximum MPC in an atomic plant or as an employee or whether he is exposed to it outside. Why do you feel that a distinction should be made?

Dr. LAPP. I think the distinction should be made on the basis, first of all, of the difference in radiosensitivity of the individuals. You are dealing with the total population now when you are dealing with the global risk. You are dealing with people who have no medical supervision. You are dealing with people who are of different ages. I believe that the International Commission on Radiological Protection recommends a factor of 10 and others believe it should be more—a factor of safety—when dealing with the total population than when you are dealing with the small population, the occupationally exposed population. These are the recommendations of the international body on the subject.

In view of the nature of our knowledge and the totality of the sample with which we are dealing, I would urge a big factor of safety in setting limits to bomb testing. It would be tragic to find someday that we had erred in setting the limits.

Perhaps I might explain, in response to Mr. Cole's question, the probabilities involved here. Supposing that the probability of damage were only one in a thousand and you only had 10 people working in the laboratory; this would be a small risk for 10 people. But if you had one in a thousand and apply the same statistics to a total population of 2 billion people, obviously, you have a very different situation. It is part of the philosophy that goes into establishing such figures.

Representative COLE. When you say, in your opinion, that we should set a big factor of safety, don't you mean that we should set a very low factor of safety in order to be on the safe side?

Dr. LAPP. Perhaps my language is not clear there.

Representative COLE. You mean the same thing.

Dr. LAPP. Yes.

Representative COLE. We should be ultracautious in fixing a factor of safety.

Dr. LAPP. Yes.

The Soviet nuclear tests: I felt that the committee might be interested in learning some miscellaneous data I picked up in Japan. I learned that the Japanese scientists collected sufficiently active samples from the Russian tests to perform radiochemistry upon the bomb debris. I was informed that five Soviet tests produced a fallout on Japan from which scientists measured and identified the presence of radioisotope uranium 237. The Soviet explosions were characterized by such fallout that they were judged to be in the megaton range. These estimates are subject to considerable uncertainty, but one authority told me that he estimated that at least 2 bomb yields were in the range of 10 megatons.

Two Soviet nuclear tests were observed to originate in the Arctic region, whereas the remaining tests took place in a region estimated to be the Ozero Balkash, which is southeast of the new coal area of Karaganda. The air-mass trajectories from central Siberia frequently sweep across the islands of Japan, especially Hokkaido. They also produce tropospheric fallout over the United States, as well. Here in Washington you could swipe a Kleenex over a car top and cause a Geiger counter to respond. Perhaps that is a qualitative statement, because the normal counting rate of the counter is 20 counts a minute, and the counter may go up several times over that. But it was readily detectable by even such a simple analysis as this.

Representative VAN ZANDT. Dr. Lapp, that last statement you make; is that a fact or is it just hearsay?

Dr. LAPP. I was told this by one of my scientific colleagues, since I was not here at the time. I have no reason to doubt it.

Representative VAN ZANDT. Then we are getting it secondhand.

Dr. LAPP. Secondhand, but I can give you the source, if you wish. I have seen the measurements done myself. The presence of uranium 237 in the Soviet fallouts proves that the Soviets have achieved a compound fission-fusion or so-called multiple-stage weapon. According to my information, this was first accomplished in September 1954.

Representative COLE. First accomplished where?

Dr. LAPP. In the Soviet Union. The next statement has already been made.

I would like now to talk about the problem of local fallout. I am not going through all of section A, because what I am doing there is trying to explain a term which I coined some time ago in order to eliminate confusion in popular translation with the megacurie. The term I used is the "eternity roentgen" per square mile. It turns out that this particular compound unit is extremely easy to use in estimating the roentgen exposure of people in a bomb area. I will not go through all of this.

Could I have the chart of Dr. Shafer put up? (See p. 119.)

Last week Dr. Shafer testified before this committee about an attack of 2,500 megatons of bomb yield upon the United States, and specified that they were surface burst dirty weapons. By dirty it is meant that the ratio of fission to fusion is high. I assumed that 2,000 tons of fission products deposited locally. This I calculate as 12 billion roentgens per square mile.

Representative COLE. You have characterized these as dirty weapons, Dr. Lapp. How would you evaluate the content or force of a weapon which might be called a clean weapon? As you know, there

has been a considerable discussion of the maining of the words dirty versus clean, clean versus cleaner. You have used the words "a dirty weapon." Are there degrees of dirtiness?

Dr. LAPP. Yes. I would estimate the degree of dirtiness as the ratio of the fission to the fusion release in the bomb.

Representative COLE. So that in your opinion it is possible to fabricate a weapon which is clean.

Dr. LAPP. Relatively clean. To answer the question precisely, the question would be whether or not you could fabricate a weapon in which there were no fission products.

Representative COLE. You could not do that.

Dr. LAPP. This I do not believe is possible. So you can fabricate a cleaner weapon.

Representative COLE. Would you conclude that it is possible to fabricate a weapon which is so clean that dirtiness is not of great importance?

Dr. LAPP. I do not have the facts on which to answer that question.

Representative HOLIFIELD. No one else has come before us who had the facts to answer that question. Dr. Graves answered the exactly opposite. He said there is no such thing as a clean weapon. There are varying degrees of dirtiness.

Dr. LAPP. If I may muddy the water a little more, I would say that one must also include here in this argument about clean and dirty bombs the operational aspects of the weapon. You have first the problem of how much dirt is actually produced by the bomb, and then you have second the problem of how much dirt comes down. If you test the bomb at high altitude and set it off at high altitude, then you minimize the local fallout.

So we have two problems here. To get an index of dirtiness of an actual weapon tested you have to apply some formula here for the eternal dirtiness, and then the operational dirtiness. So it is a complex thing.

Senator ANDERSON. Doctor, if you had developed a type of bomb that would not explode high in the atmosphere but equipped it to explode close to the ground, would you add or subtract from its dirtiness? Would you not add to its dirtiness, so-called, to get down where it picked up particles of soil?

Dr. LAPP. When you pick up, I would call it the ballast, the soil debris, it tends to maximize the local fallout and make the weapon dirtier.

Senator ANDERSON. Therefore, if we were to prove whether our interest was clean or dirty weapons, we would need testimony from the military as to whether they had or had not developed weapons that would explode closer to the ground.

Dr. LAPP. I believe that the operational aspects, namely, the altitude of detonation would be very important. The testimony that Dr. Shafer gave is illustrated on this chart in which the varying degrees of contamination are illustrated by different colors. I am not going to use the exact figures. I merely wanted to illustrate the type of continental contamination that you get into if you have an attack with 2,500 megatons of dirty bomb.

I would take an example and then discuss the implications of dirtiness in a strategic attack upon a country. If you assume that 50 percent of this dirtiness falls out locally, you have then 1,000 megatons

of fission products concentrated upon Northeastern United States, the region which I use, and much of the region would then be subject to a fallout of 10,000 eternity roentgens.

May I define that? The eternity roentgen is a unit of the exposure from one hour, considering that the time of fallout to deternity, it is divided up on the following time schedule. May I jump to section C, which is the persistence of fallout. May I bypass the comments about this and jump directly to the data.

If you have 10,000 eternity roentgens, this amounts to a rate of 2,000 roentgens per hour at one hour. Here is the time table of the delivery of the roentgens to a person exposed in the open on a flat area. From the first hour, assuming the fallout occurred, then, to the end of the first day, there would be 4,700 roentgens of exposure. That would be about 10 times the lethal dose for an individual. So he had better be somewhere besides the top of the earth.

From the end of the first day to the end of the first week, there would be a dose of 1,730 roentgens, or about 3 times the lethal dose. From the end of the first week to the end of the first month there would be an additional dose of 920 roentgens which would probably also be lethal. From the end of the first month to the end of the first year, there would be slightly over 1,000 roentgens. From the end of the first year to 50 years would be an additional 840 roentgens, but I make the note that weather and terrain would make a significant difference in cutting down that dose.

This point, I think, is worthy of stressing, because of its great implication for civil defense and for analyzing what the ultimate consequences of an attack upon a country are. You have the problem here of confronting 920 roentgens from the first week to the end of the first month after the attack, and even after that is over, you have the problem of the 11-month dose of slightly more than 1,000 roentgens. After the first year, there would be a smaller dose of some 840 roentgens that would be the theoretical maximum.

Having listened to Dr. Crow's testimony yesterday, I proceeded to calculate late last night or early this morning—I forget which—just what this would mean because Dr. Pollard and the others pointed out the consequences of an attack upon the United States, and Dr. Libby this morning emphasized the great consequence of a nuclear attack itself.

Representative HOLIFIELD. I am glad you are going into this, because the chairman has received any number of telegrams—in fact, 1 was delivered at 6 o'clock this morning when my doorbell was rung by a messenger, and they got me out of bed to give it to me—condemning the committee for not going into the effects of a war and possible multimegatons. Apparently the people writing in are not aware of all the testimony, and are not aware of the fact that the extrapolation from this information can be applied to multimegaton exposure of the population. So I am glad you are bringing that point out.

Dr. LAPP. I just had the opportunity of discussing this at lunch with Dr. Crow. I have some adjustments to make with it, but nonetheless in view of the nature which he will agree is fairly approximate data, I will let these figures stand.

What I did here was the following: I assumed that by some means we were extremely fortunate in having the attack. I mean we were

fortunate after the attack in being able to hide our people from acute dosage during the first month.

I went on to calculate how much dosage these people would get if we took a generalized smear out of the radioactive fallout over the continental United States. Here is where the eternity roentgen square mile is an extremely useful concept, because I can simply make the calculations very quickly.

What I did was to arrive at through this mathematics adjusting for weathering at an average dose of 400 roentgens for the average exposure to every American who survived an attack of 2,500 megatons, with some 2,000 megatons of fission products released.

Senator ANDERSON. Doctor, it is hard to translate these things back and forth. The other day when Dr. Russell was testifying, we got into the question of the fact that if a person was exposed to so many units, it might shorten the lives of children by a certain number of days and so forth. Are these roentgens what you have called eternity roentgens the same thing he was talking about?

Dr. LAPP. I have adjusted. I used the eternity roentgen just for the simplicity of calculation. I have gone back to the pure roentgen.

Senator ANDERSON. 400 roentgens would be a very substantial dose for not only the person exposed, but would have very, very substantial effects upon children that generation and continuing, as he pointed out, for several generations to come. Geneticists did not stop when the individual was exposed. They went right along through the several generations.

Do I understand that this is a sufficiently large dose so that it would shorten the lives of those children depending upon whether you used the upper or lower limits of Dr. Russell's table from 10, 15, or 20 years or something of that general nature, if the father and mother got 400 roentgens.

Dr. LAPP. I am not familiar with Dr. Russell's data. I am sorry.

Senator ANDERSON. Are these the same roentgens he was thinking about?

Dr. LAPP. They are the same roentgens. The roentgen is the roentgen and if he was talking about the roentgen, this is the same roentgen. If he was talking about the neutron unit, this would be different.

Representative COLE. Was he talking about the neutron unit?

Dr. LAPP. As I judge from what I heard in this hearing, I thought he was testifying about the neutron unit. Is Dr. Russell here?

Representative PRICE. He definitely stated he was talking about the neutron.

Dr. LAPP. You all saw Dr. Crow's table, I believe, showing the expected genetic effect upon the first generation and upon the total succeeding generations. What I did was to calculate how this would scale up if you had an aftermath of a nuclear war under this very optimistic condition that the people who survived got no radiation for the first month, but then were exposed to a cumulative dose of 400 roentgens over the period of a generation—a reproduction generation. The first generation, according to this table, would have 1,600.000 physical and mental defects, a total of 16 million for all total generations. There would be stillbirths and childhood deaths of 4 million, a total of 120 million for all generations. Embryonic and neonatal deaths, 8 million, and 140 million total, and a much larger but unknown number of intangible defects. (See p. 1021.)

I think I would have to change this on the basis of what Dr. Crow told me, instead of 2 out of every 10 children in the United States in the first generation would be genetically defective, it would have to be 1 out of every 10. The sum total of all deferred deaths from the attack would be 272 million. I believe that should be scaled down because of what he told me to somewhat less than that. I believe about 150 million. I will have to check with Dr. Crow on this.

This, then, is the kind of genetic consequence from an attack which, according to Dr. Shafer, would have produced, I believe, of the order of 80 million deaths.

This kind of a calculation is to me a rather awesome one. When I was thinking it over and talking with Dr. Crow at lunch, I was thinking, supposing this happened and you tried to imagine what you could do about it in advance. Of course, one of the things you could do is to arrange for shelter of people, but shelter of people for the time periods we have in mind is going to be increasingly difficult even if we have the funds for it. I hope I can be forgiven for injecting in the testimony at this time a thought which I had. It is the nature of nuclear warfare which provokes this. There perhaps might be a national objective to have a stockpile of human sperm—the male sperm—which would be stockpiled at strategic locations in the United States for providing at least on the masculine side a pure line of nonirradiated sperm. I realize that this may seem like a bizarre suggestion. I understand according to biologists that you can keep human sperm viable for considerable periods of time. If you did that, then I believe you would cut your genetic consequences more than in half, because I understand that the female is less sensitive to radiation than is the male in terms of the sperm versus the ovum. This means you could cut in half or less than half—you could probably cut down between a half and a third—the consequences to future generations. It may even be, and here we would have to do a great deal of research, if you could continue the integrity and viability of the sperm through more than one generation, you then could continue nonirradiated nonmutated sperm through more than one generation.

I realize here I am getting a little fanciful. I am merely injecting this into our discussion, the kind of things you come up against when you consider the awesome consequences of nuclear warfare.

Representative COLE. Dr. Lapp, you have, of course, posed a most intriguing and bizarre as well as fanciful suggestion, but it occurs to me that is it not likely that if such an event occurred in which such a large proportion of our population were affected to the point where it would be advisable or helpful if we could have a reservoir of sperm, would not that concentration also affect other animal and plant life to a degree in which even though we were able to reproduce the human race, we nevertheless could not survive because of insufficient food, water and such?

Dr. LAPP. Mr. Cole, I believe that so far as the crops reproducing themselves are concerned, this would not be the fundamental problem, because I believe the mutation rates are quite different in crops and some of the other animals. I would like the specific question of the relative biological effect genetically to be addressed to one of the geneticists.

Representative HOLIFIELD. Of course, the suggestion you have made is an unusual suggestion, but we are dealing now with a world in

which the possibility of releasing these quantities of megatons of fission are either here or will be here very soon. It just accents the gravity of nuclear warfare and this is, of course, one of the things which mankind has to deal with for survival of the human race. If we are going to have this kind of warfare, these are the problems that are presented.

Dr. LAPP. Mr. Holifield, I personally believe that projections of the probable consequences of a nuclear warfare are in themselves the greatest deterrent to war. But this has to be absorbed on both sides of the Iron Curtain. From what Dr. Muller said yesterday about the state of genetics in the Soviet Union, I think it might be quite advisable to make sure that no one in the Soviet Union is in doubt as to the consequences of a nuclear war.

I am at a loss to say how to do this, but it might be accomplished through a good conference on genetics to which the Russians were invited.

Representative COLE. Dr. Lapp, you have indicated that since your luncheon visit with Dr. Crow you have revised your conclusions from your original script which estimated an effect on the first generation of 2 out of every 10—since your luncheon visit with Dr. Crow you have revised that downward to 1 out of 10, which is a very striking revision, a difference of a hundred percent; if that can be the consequence of a luncheon visit with Dr. Crow, might it not be conceivable that if you spent a dinner evening with Dr. Russell and other scientists, you might further revise your figures one way or another, or if you spent a week with them there might be even a greater revision?

Dr. LAPP. I am not sure that the degree of revision would be proportional to the time of contact with these individuals.

Senator ANDERSON. You would find out that at one time the Atomic Energy Commission had a figure of 50 which in a short time they brought down to 2. Maybe they should go to dinner also.

Dr. LAPP. I apologize to the committee for introducing this figure, but I did not understand from Dr. Crow's testimony yesterday that these two figures he gave were not mutually exclusive.

Representative HOLIFIELD. Mutually what?

Dr. LAPP. The point was that in the column of data he presented, he had two figures, one of which actually enveloped the other. I had thought they were separate. I was in error.

Representative COLE. I was impressed by the apparent fact that a casual luncheon conversation could result in such a striking revision of your conclusions.

Dr. LAPP. The same factor of two would have been produced from a single sentence he gave me when I showed him the results. This is entirely in the nature of how scientists iron out these differences. They talk with one another.

Representative HOLIFIELD. I might say that we have had many statistics given to us that range all the way from a factor of 2 to a factor of 10 or 15. So this variability in your figure is not unusual to other testimony we have had.

Dr. LAPP. I believe in presenting the data Dr. Crow mentioned that it probably was not exact by a factor of three. I suspect it could be even more. I am merely using this as an indication. I would claim no precision.

Representative COLE. But his admission that his calculations might be in error by a factor of 3 was based upon long periods of sober study and concentrated thought, and that even after such long period of study, he came to certain conclusions which he admits might nevertheless be in error by a factor of 3.

You have indicated that your statement today was composed in the wee hours of last night and the early hours of this morning, as well as the luncheon visit. Therefore, might it not be reasonable to conclude that your estimates might be in error by a factor of as great as 15 or 20?

Dr. LAPP. I think the physical calculation I have made I would be willing to stand on. I think it is correct within the method that I have estimated the figures are accurate. The uncertainty comes in when I apply it to the genetic data. Actually, I think, Mr. Cole, I have underestimated the situation deliberately. I have taken lower values, not upper ones.

One of the most important things that I wanted to discuss at least in my opinion before this committee, and I hope I can jump to it is the problem of the present test rate. I have indicated in the last page of this testimony my rough estimate—and I emphasize that it is a rough estimate upon all the data available to me of the rate of testing, the injection of fission products into the stratosphere over the past period of time since the first bomb was exploded in 1945.

This is a semilogarithmic plot. That is, the scale on the left in logarithmic. Starting at the bottom, the lowest value given in one megaton, and it runs up to 10 and 100 megatons of fission products injected into the stratosphere. To be perhaps redundant, may I explain that this is the number of fission products associated with the explosion of 100 million tons of TNT equivalent fission energy in a bomb. The reason why nothing appears up in 1951, up to the small value which I indicated as two-tenths of a megaton, is that prior to that time we were in the fission domain of weapons—the pure fission domain of weapons—in which the weapons had their energy released by a chain reaction in fairly expensive uranium 235 or plutonium 239, material which, as a rough estimate, we can say cost $10,000 a pound. The price is somewhat less now. Up until that time we were dealing with relatively small weapons and because of the fact that these weapons did not have great explosive power as compared with the megaton class weapons, their fission debris was restricted to the lower atmosphere or to the troposphere.

This meant that so far as the global aspects of radioactive contamination were concerned, and considering the method of transportation, the total amount of fission debris deposited was small and negligible. It was only when we entered into the era of the megaton in weaponry that we started to get into a situation where the injection of radioactive fission products into the stratosphere because of consequence and could be measured remotely all over the world.

If we look at that, we will find in the injection corresponding to the test in 1952—by the way, this includes the United States and U. S. S. R. I have not included any contribution from the British—and in 1953, relatively little testing so the curve goes down. In 1954 was when we had the Castle series of tests with a total estimated contributed fission yield of 30 megatons into the stratosphere.

The next year was cut back to a total of 3 megatons and then the next year, 1956, it went up. If you will permit me to go to another curve, I can give you the estimate. It is to about 14 megatons. I am not in a position to make an estimate of the 1957 contributions since I am not aware of what the British have contributed with the Christmas Island test, and am only partially informed of what the Russians have contributed with their spring series of tests. But I believe that this chart does illustrate some of the problems you have in discussing the present rate of testing.

One way—and this is the way Dr. Langham suggested—is that you simply average the testing over the past 5 years, which would mean we take these 5 bar graphs and add them up and divide by 5.

I believe that when this is done that this curve will come out to somewhat more than Dr. Langham estimated. I am not sure that in just a minute I can give you the answers to that.

Senator ANDERSON. Very close to it.

Dr. LAPP. It is close to it.

Representative HOLIFIELD. Of course, this excludes the Russian test.

Dr. LAPP. This includes the Russian test. I have been conservative with regard to the Russian test, probably overly conservative, because of the problem of estimating just what fission yield they had in the weapons, how much was injected into the atmosphere, and how much dumped out stratospherically.

Senator ANDERSON. Dr. Langham said it came to about 50 megatons in the past 5 years. If you add your figures, it comes out to about 50 and a fifth of that is 10 megatons per year.

Dr. LAPP. It is roughly 50, sir. I do not have the complete detail here as to how I arrived at all these figures. The reason for the Castle series of test figures being there is due to Dr. Libby. He presented the data which allows me to derive this value for the Castle series of tests. That is the principal contributor, and thus explains why my estimate should be so close to Dr. Langham's.

There is one thing which has puzzled me, that is, that if these data are in fact correct, then the Russians have not really tested a series comparable to our Castle series of tests.

Senator ANDERSON. It could be that we had one fairly sizable shot that has not yet been approximated by anybody else. That is a possible explanation.

Dr. LAPP. That is possible. I can only draw an inference.

Senator ANDERSON. Would you not be satisfied to take the total figure that Dr. Langham used in view of the fact that he is so closely associated with Los Alamos Laboratory which for many years of this period was doing the major part of the testing and has since been joined by Livermore—but the two laboratories work very closely together and their scientists are certainly knowledgeable of everything that has taken place in the tests thus far—they have the translations of what the Russian tests have been insofar as we are able to detect them, and could you not agree with him that 50 megatons would be about the total for 5 years and 10 for each year?

Dr. LAPP. I believe the agreement is very close, and I would certainly go along with Dr. Langham. The range of estimates for the test limit for the injection of radioactive material, assuming equilibrium, runs in the range of from 2 to 10 megatons, according to the discussion before this committee at the round table of Dr. Neuman and Dr.

Langham, Dr. Eisenbud, Dr. Kulp and one other person whom I have forgotten.

Because of the way I have made estimates before, I have used a value of 3 megatons per year, with a factor of 3 either way. In other words, it might be as low as 1 megaton per year or it might be as high as 9 megatons per year. One can see from this that our own test rate has exceeded this value. If we take three, it is exceeded about twice. If we take 10, it has exceeded once plus the Russian contribution, which would be a global passing limit of 2 times in the past decade.

This is assuming equilibrium. You are going to have to have more tests before you will load up the stratosphere. So from the standpoint of being reckless, the United States has not yet exceeded the limit.

I would like to make that very clear. I do not think my written statement adequately brings that out.

I realize I am taking a great deal of the committee's time, and I would like to go through some of my testimony relatively quickly. Perhaps the question of the future nuclear tests should be discussed briefly. I think in dealing with future commitments of radioactive debris to the earth's atmosphere we must deal with many unknowns. Had we attempted an estimate 5 years ago prior to Castle-like weapons we would have arrived at most misleading and optimistic projections. The end of weapon development is not in sight, and no one can say that unexpected developments may not occur. For example, may not smaller nations be stimulated by British success with thermonuclear type weapons and place maximum emphasis upon such development?

Additionally, can we be sure that a nation would restrain itself and not test a 100 megaton dirty weapon if military requirements and nuclear technology indicated that such a weapon was desirable in its own security interests.

Will not the requirements of adapting maximum megatonnage to a small warhead put emphasis on further development of dirty weapons?

I cannot answer these questions at this time, but I know that single weapons tests of very high fission yields can add a strontium burden to the atmosphere far beyond the limits we have been discussing here.

The United States has contributed the largest fraction of the radio strontium to the stratosphere, and I think it is distinctly encouraging that the fullest discussion of the strontium fallout should occur in this country. I am not aware of any large body of published information on this subject of Soviet origin. It is known, however, that the Soviets are engaged in strontium studies.

In concluding this section, I would again like to stress Dr. Libby's contributions to this subject. They are of very great value and I feel sure that we would be in a much poorer position today to evaluate the strontium problem were it not for Dr. Libby's personal interest in this field of investigation and the research which he has pushed so vigorously.

On the strontium problem itself, I would like to state briefly strontium 90 determinations in man must be expanded to assess the increase in strontium 90 burden which will occur in future time. Careful determinations of natural strontium in humans deserve increased

attention. We know that more strontium 90 will accumulate in humans as a result of bombs tested in the past and as a result of current tests. I believe that Dr. Selove is going to give a further discussion of this. The determinations as to how much of the radio element may be tolerated safely is a matter for the biologists to discuss.

This committee has heard a fairly wide range of opinion from its expert witnesses on the probable biological effects of strontium 90 in man. But it seems to me that even in this area some agreement was reached, especially when Dr. Shields Warren stated on June 3:

I would be reluctant to see the average strontium 90 content of bones, particularly in children, go much above 10 times the present level.

Dr. Libby's speeches show that the strontium 90 fallout will continue, and the strontium 90 level in human bones will increase.

I believe that unless restraints are imposed upon commitments of fission products to the atmosphere, it is only a matter of time before the strontium 90 level of Dr. Warren will be reached.

I would not be able to extrapolate that curve very well into the future to determine this.

I would like to jump to constructive proposals—at least I believe they are constructive—if I may just insert one comment here.

A colleague, Dr. Jack Shubert, who is presently at the Laboratory for Inorganic Chemistry in Zurich, Switzerland, from the Argonne National Laboratory, and he has been in Britain recently and discussed data with the British on the question of the relative sensitivity, I would like to quote from a letter I received last night from Dr. Shubert:

It used to be thought that at least 1,000 roentgens of absorbed radiation would induce cancer. Within the past few years, it has been found that as little as 200 roentgens delivered to children would induce cancer in later life. Now it has been found (this is by the British) that as little as 3 to 5 roentgens received by the unborn child in its last 2 months before birth has been responsible for cancer of all types appearing a few years later.

I believe that this statement from Dr. Shubert which represents the final conclusions of the data of Dr. Alice Stuart in England, is significant in that it does show that the incidence of cancer malignancy in children correlates with the X-ray of women prior to term. This is diagnostic use of X-rays which in the case of X-rays may involve of the order of a few roentgens, an amount which was thought harmless.

I believe also that this would have significant bearing upon the question of the threshold.

In order to allow time for my friend, Dr. Selove, may I jump quickly to my proposals and read them for you.

A. ATOMIC ENERGY COMMISSION INFORMATION POLICY

I suggest that this committee or its parent committee may wish to review the information policy of the Atomic Energy Commission, and I might add the Defense Department, with regard to nuclear weapon effects, with a view toward revising this policy so that information may be made available more promptly and completely. I am thinking particularly of the relations of the Atomic Energy Commission with the press. I believe that the national interest de-

mands a better relation, a freer flow of communication between the Atomic Energy Commission and the press.

My second proposal, I suggest that the Joint Committee on Atomic Energy might wish to recommend or to sponsor the preparation of an analysis of the probable biological effect of nuclear warfare. It would be useful to investigate probable lashback effects from various levels of nuclear bombardment. What I am thinking of under this category of lashback is the fallout which would occur upon the country which uses the nuclear weapon itself. That would be the remote tropospheric fallout.

The third point, data useful to civil defense. I believe that the committee's investigations have produced information of critical value to the civil defense planning. It might be useful to have a summary report of these data transmitted to the FCDA. I say that because I personally have not seen many representatives of the FCDA at these hearings.

Research in long-range estimation of nuclear explosives. It is known that considerable effort has focused on long-range detection of nuclear detonations. Attention should be given to the declassification of such data as would bear upon evaluation of the radio strontium problem. In particular I have in mind helping us to estimate how much the Russians are contributing, if this can be done, without jeopardizing sensitive data. Other data would be most useful in discussion of the feasibility of policing an agreed upon test limit, if one could get a multilateral agreement.

Annual fallout report. In view of the great public concern over fallout hazards, I would urge that the Atomic Energy Commission be required to issue an annual report on the degree of fallout and its uptake in biological systems. Perhaps the Atomic Energy Commission might wish to have that report prepared by a university task force.

Finally, I would urge that the Congress continue its investigations of radiation hazards, extending them into the broader area of peacetime uses of radiation. I believe that the ever-increasing uses of radiation must be subject to legislative controls. Radiation protection in the United States needs, in my opinion, uniform legal status.

Representative HOLIFIELD. Thank you very much, Dr. Lapp. Unless there are questions, we will hurry on to our next witness, Dr. Selove, in order that we might finish with him in time for the conference.

Representative COLE. Mr. Chairman, I should like to ask a few questions of Dr. Lapp, in order only to clear up what appears to be a conflict or some discrepancy in statements with respect to his experience and connection with the atomic energy program.

Dr. Lapp, have you seen the biography of yourself which has been prepared by the Joint Committee?

Dr. LAPP. I am sorry, I did not.

Representative COLE. I wish you would look at it if you would to see if that is a correct representation of your activities in the field of atomic energy.

Representative HOLIFIELD. I understand that the staff took that from the American Men of Science compilation.

Dr. LAPP. I believe it is correct from cursory examination.

Representative COLE. I would call your attention particularly to that part of the biography which states that you were Deputy Execu-

tive Director of the Atomic Energy Commission Joint Research and Development Board during 1947–48, and Executive Director during 1948–49. The information which has been given to me from reliable sources is the fact that you have never been an employee of the Atomic Energy Commission.

Dr. LAPP. That is correct.

Representative COLE. That is why I am giving you an opportunity to clarify this apparent discrepancy.

Dr. LAPP. May I read what it says here? It says, "Deputy Executive Director, Atomic Energy" and then there is an unusual abbreviation "Cmm Atomic Energy Committee of the Joint Research and Development Board," which was part of the Defense Department. It was Vannevar Bush's show. I am happy you brought this up. Frequently (I give lectures) I am introduced in a way which is embarrassing to me because it is incorrect. I have been introduced as everybody from the Chairman of the Atomic Energy Commission on down the line. This statement actually is correct, if you understand that is committee and not commission.

Representative COLE. How can you possibly get committee out of "Cmm"?

Dr. LAPP. I am not responsible for this. I am sorry.

Representative COLE. Then at any rate it is a fact that you have never been an employee of the Atomic Energy Commission or a consultant to the Commission.

Dr. LAPP. That is correct, Mr. Cole, I have never represented myself as such.

Representative COLE. I am not saying that you did. I am simply trying to give you an opportunity to clarify the facts of your experience in this field.

Dr. LAPP. I am glad to have this opportunity to state on this record that is correct.

Representative COLE. I further call your attention to the item "Head, Nuclear Physics Branch, Office of Naval Research, Department of Navy, 1949–52."

Dr. LAPP. That is incorrect.

Representative COLE. That is incorrect?

Dr. LAPP. That is incorrect.

Representative COLE. What is the fact?

Dr. LAPP. It was Acting Head, Nuclear Physics Branch, Office of Naval Research, Department of the Navy, 1949.

Representative COLE. 1949?

Dr. LAPP. I am sorry. Again I am not responsible for that.

Representative COLE. It is the fact that in 1949 you resigned from that post with the Office of Naval Research.

Dr. LAPP. That is correct.

Representative COLE. I do not press you on it, but would you care to give your reasons for your resignations?

Dr. LAPP. I stated on the record that I resigned of my own volition at that time.

Representative COLE. I am sure you did of your own volition. Were there peculiar circumstances surrounding the resignation?

Dr. LAPP. I had taken the job with the Atomic Energy Commission—pardon me, you have me mixed up—at the invitation of Dr. Widdel as a temporary appointment while he slid out from the posi-

tion of Head of the Nuclear Physics at that time. I took it for a short time and then resigned, I think, in June of 1949, Mr. Cole.

Representative COLE. Then there were no unusual circumstances that prompted your resignation?

Dr. LAPP. I think I would have taken this job only for a short time. I was lecturing or beginning to lecture. I was finding it difficult to lecture and also be employed by the Defense Department. But the job was temporary.

Representative COLE. That is all I have, Mr. Chairman.

Dr. LAPP. May I read the rest of this to make sure there are no other corrections? I believe it is correct as it stands now, as corrected.

Representative VAN ZANDT. Mr. Chairman, there still is in the record this statement attributed to Dr. Lapp, "reckless or nonsubstantiated statements do a disservice to the AEC and to the Nation." This statement was directed at Dr. Eisenbud, Dr. Libby and Dr. Doan. I would like to ask the Chair if we are going to give these three distinguished Americans an opportunity to answer the accusation?

Representative HOLIFIELD. Yes, sir. Would the committee like to have them come forward at this time or would you like to go forward with the next witness and have that take place in the conference?

Representative COLE. Mr. Chairman, if we are through with Dr. Lapp, I would strongly urge that we invite Dr. Eisenbud, who is in the audience, to come immediately to respond to Dr. Lapp's charge.

Senator HICKENLOOPER. Mr. Chairman, I would suggest that we finish with Dr. Lapp first. I have some questions I would like to ask.

Representative HOLIFIELD. Senator Hickenlooper has some questions, Dr. Lapp.

Senator HICKENLOOPER. Dr. Lapp, have you ever done what might be called extensive works in genetics under your own responsibility in connection with the effects of radioactivity on human cells?

Dr. LAPP. No, sir.

Senator HICKENLOOPER. Have you ever done any research in biology and medicine in connection with the effect of radioactivity on the genes or other parts of the human body?

Dr. LAPP. I have never done any research on genetic effects.

Senator HICKENLOOPER. Have you ever conducted any research yourself on the fallout or its intensity in this country? By that I mean any laboratory research of any extent.

Dr. LAPP. I have made some simple measurements myself, but my data here and my testimony is based upon the data of the Atomic Energy Commission.

Senator HICKENLOOPER. That was the next suggestion that I wanted to make. I have the impression here—I do not know whether I am correct or not—that you are appearing here more in the nature of a reporter or a correlator with some considerable educational background, I admit, of the scientific data as you personally interpret it, which has been compiled by a number of eminent scientific people who have actually done the work.

Dr. LAPP. I believe the invitation that was extended to me by the committee more or less put me on these lines of testimony.

Senator HICKENLOOPER. Yes. The only purpose of this suggestion is that you are not here, while you are a scientist in your own right on your education, giving firsthand evidence based upon data with which you have been acutely or intimately connected in connection with its

development. So you are reporting what you have read or what you have been told by others with varying degrees of accuracy and drawing your own conclusions.

Dr. LAPP. Mr. Hickenlooper, I think it is a fair statement to say that I have done considerable work based upon information made available by the Atomic Energy Commission and scientists in general. I have done active work in gathering data from people, especially as every scientist will do, when available.

Senator HICKENLOOPER. I know that. I make the point that there are several very eminent newspaper reporters in the scientific field who have also done a tremendous amount of work of gathering data and yet they have done no research of their own. They are reporters and in a very proper field they are reporting what they find. I merely have the impression that you are here today in the nature of a reporter or a compiler of information which you are interpreting as you see it. As differentiated, I might add, from the testimony of most of the rest or all of the rest, perhaps, of the witnesses we have had here who have been intimately working in this field and present the results in the main on their own experience, and the results of their own work.

Dr. LAPP. I believe, Mr. Hickenlooper, this is within the framework of the outline which requested me to testify.

Representative HOLIFIELD. I am very sorry. In order to correct the record, the Chair will have to state that Dr. Libby is a chemist, and the information he has given us in every field of science has been obtained from his association with other scientists and reading of other materials. So in order to keep the record straight, let us have the facts spread there that it is not only Dr. Lapp who had studied the other scientists' work and reported on them, but also Dr. Libby, one of the AEC Commissioners.

Dr. LAPP. May I say in general response, not specifically to Mr. Hickenlooper, that I admit to being critical of the Atomic Energy Commission. My criticisms of the Atomic Energy Commission, I always felt, have been directed toward trying to bring the facts out into the open, and the free play of public discussion. As Dr. Libby testified this morning, some of the problems involved here transcend the area of science. I have felt that this is important. I have tried to help in the somewhat new but I think important problem of education in science.

Senator HICKENLOOPER. Then where do you obtain your facts? Do you have access to restricted data?

Dr. LAPP. I have no access to restricted data, Mr. Hickenlooper. The facts that I have presented here are based upon information which is available freely in the scientific domain.

Senator HICKENLOOPER. Not only in the scientific domain, but from the Atomic Energy Commission also. Is that not true?

Dr. LAPP. Such data as the Atomic Energy Commission published. We were assured this morning by Dr. Libby that all of the data except for a very small fraction dealing with a long-range detection on Project Sunshine had been put in the public domain.

Senator HICKENLOOPER. What information do you have that the Atomic Energy Commission is not making available, if you have no access to restricted data, and if your information comes from data freely available from various sources including the Commission? Does not that indicate that data is being made available?

Dr. LAPP. If I may respond to the question, Senator Hickenlooper, my complaints in the past about the slowness with which the Atomic Energy Commission emitted data, which I thought were vital to civil defense, were, for example, that the data were not available. They had to be derived by me from other sources, other than the Atomic Energy Commission. As for example, the scientific data from Japan. Thus I think the fact that later on the Atomic Energy Commission actually confirmed through its pronouncements that these results were correct testified——

Senator HICKENLOOPER. Have you had an opportunity to go through the data in minute detail from Japan, examine the records and all the data that they have developed, or are you relying entirely upon the verbal statements of certain scientists made to you in the course of your visits with them?

Dr. LAPP. No. I have been in communication with Japanese scientists for some time. I have received letters and photostats and copies of their scientific papers, in some cases prior to publication.

Senator HICKENLOOPER. Thank you very much.

Representative COLE. Mr. Chairman, I had intended to interrogate Dr. Lapp just a little bit about his visit to Japan earlier when he referred to his visit in his statement. While I hate to keep him on the stand unduly, I am curious to have his observations on two points.

One is, Dr. Lapp, that you were registered as one of the scientists who would attend the conference.

Dr. LAPP. Yes.

Representative COLE. And yet you did not attend the conference.

Dr. LAPP. No; I did attend, Mr. Cole.

Representative COLE. It was reported to me, and the reason I raise the question is because that same question was raised by a number of persons who were there, as to the reason why you had not attended the conference.

Dr. LAPP. I would be glad to explain this.

Representative COLE. That is one point. Then the other, since you remained in Japan after the conference was concluded, I am interested to have your observations with respect to the accomplishments of the conference.

Dr. LAPP. On the first point, I arrived in Japan on, I believe, May 1, considerably in advance of the conference. It was my firm intention to attend every session of the conference, because I am very much interested in the work of the Atomic Industrial Conference. However, when I got to Japan, I found myself in a pretty mad race because I was digging up some data on the *Lucky Dragon* story, interviewing some of the fishermen who were in this unlucky boat; and I wanted to visit Hiroshima and visit some scientists in the various places. I found that when I had to deal with just the problem of hours spent in talking with the fishermen that by the time I got back to Japan the conference had already started. This was unavoidable, but I had to meet people at certain places.

Then the amount of commitments I had with the press and visiting the *Lucky Dragon* itself precluded my going to more than one session.

Representative COLE. Which session was that? It does not matter.

Dr. LAPP. I can give it to you. I did attend that. I wanted very much to attend the luncheon and dinner sessions. I had all the tickets, and I just could not use them. For example, the one night I wanted

to do that, I think I was invited to a banquet by one of the prominent newsmen. So I, unfortunately, did not have much time.

As for the second point, just what was my reaction, I did not have too much opportunity to discuss with the Japanese scientists their reaction to the conference, but I gathered from conversation with a few of them that they were greatly impressed with the conference. It was very well managed. It was well attended. I think they were very much impressed with the general conference.

Representative COLE. Then your conclusion is that it was a very worthwhile conference.

Dr. LAPP. I would say so.

Representative COLE. That is my own conclusion.

Dr. LAPP. Yes.

Representative COLE. I can also verify your statement that it was an extremely well-organized conference involving a considerable number of people. There were some meetings that were attended by as many as 4,000, 5,000 and 6,000 Japanese. It was an unusually well-organized conference, probably the first international conference of industrialists and scientists that has ever been held.

Dr. LAPP. I would in no way like to detract from the value of this conference. My only regret is that I was able to attend so few sessions. I cut short my visit in order to come back here. I would have liked to stay in Japan much longer.

Representative COLE. My only basis for concluding that you had not attended the sessions of the conference is that I inquired—because I looked forward to seeing you there—inquired at the registration desk at the end of the conference, and they said you had not, so far as they knew, attended the conference, and the papers in the box—there was a slot for each of the delegates—were still there.

Dr. LAPP. I have the papers in my office, Mr. Cole. Again I regret, and no one does more than I, that I could not attend these conferences. I was working. The press in Japan can at times be very aggressive. Perhaps you found that out yourself.

Representative COLE. Yes.

Dr. LAPP. I found myself going to a 15-minute interview and ending up with a 2-hour luncheon.

Representative COLE. Now, with respect to your sources of information, Dr. Lapp, you have indicated that your observations back through the years are based on reports and studies of unclassified data that have come to your attention. Are they not, also based on discussions with other newspapermen and analysts in this field, such as yourself?

Dr. LAPP. I am proud to say that I communicate with a great many members of the press.

Representative HOLIFIELD. Mr. Eisenbud, you are now given an opportunity to reply to the comment of your colleague in science, Dr. Lapp.

Senator ANDERSON. Before you do so, Mr. Eisenbud, may we sort of follow along in this same pattern of qualification? I look at your qualifications in here, and I see "EE, New York University." Does that mean "electrical engineer"?

Mr. EISENBUD. Yes.

Senator ANDERSON. What subsequent degrees have you acquired?

Mr. EISENBUD. I have no subsequent degrees.

Senator ANDERSON. Just the degree as electrical engineer?

Mr. EISENBUD. Many years ago; yes, sir.

Senator ANDERSON. I see you spent more time working for the Liberty Mutual Insurance Co. as a hygienist than anything you have done in your life and more than all the rest of your experience put together; is that right? Eleven years with them, and only 10 years since then.

Mr. EISENBUD. That is about right.

Senator ANDERSON. I am not trying to be critical, because I tried to speak appreciatively of what you had done a few moments ago. As a hygienist, did you ever have occasion to get into the question of deposit of strontium 90 in the atmosphere for the Liberty Mutual Insurance Co.?

Mr. EISENBUD. This was previous to 1947. Up until that time very little strontium 90 had been formed in this world. The radiaition problems of those days were X-ray and radium. We were only beginning to get interested in the kinds of things which we are talking about today.

Senator ANDERSON. The field that you were in had to do with the detailed discussion of the occurrence of strontium 90 and cesium 137 in the atmosphere, biosphere, and its uptake and behavior in man. What was there in your electrical engineering course that dealt with that?

Mr. EISENBUD. Very little, sir. But a great deal in some 21 years of professional experience, during which time I have attained the rank of adjunct professor of industrial medicine at New York University Medical School.

Senator ANDERSON. What was there in this work with the Liberty Mutual Insurance Co. as a hygienist that started you off lecturing on medicine?

Mr. EISENBUD. I got interested, about 22 years ago, in a legitimate subject for a young electrical engineer, namely, the electrical charges on dust, and went from there to the general physical properties of dust, and then into the physiology of dust and dust diseases, and spent a great deal of time from 1936 until 1947 studying the general behavior of dust, not only in the atmosphere, but in the lung and in the body.

Senator ANDERSON. Your paper was headed, "A Measurement of Strontium 90 in Geophysical and Biological Material." Are you a geophysicist?

Mr. EISENBUD. Sir, I do not know, really, how to answer that. I think anybody that has some interest or qualification in geography and geology and some in physics could at least write on the subject of geophysics, but I am not a geophysicist.

Senator ANDERSON. Are you a biological worker.

Mr. EISENBUD. I work in the biological field. I, myself, am not a biologist.

Senator ANDERSON. Have you conducted experiments in this measurement of strontium 90?

Mr. EISENBUD. I have directed the experiments, and have conducted some myself.

Senator ANDERSON. Have you conducted them? Have you done any experimental work yourself?

Mr. EISENBUD. The experimental work was performed under my immediate supervision. I have done some of the experimental work myself.

Senator ANDERSON. Most of it was done under your supervision?

Mr. EISENBUD. This has been a large program. This is much too large for one man. It was done by my immediate staff.

Senator ANDERSON. Then you are here as a reporter of what someone else has done.

(A letter from Merrill Eisenbud, setting forth a full record of his qualifications, follows:)

UNITED STATES ATOMIC ENERGY COMMISSION,
NEW YORK OPERATIONS OFFICE,
New York, N. Y., July 12, 1957.

Mr. HAL HOLLISTER,
Staff Member, Joint Committee on Atomic Energy,
Congress of the United States, Washington, D. C.

DEAR HAL: You will undoubtedly recall that during the proceedings of June 5, the question of my professional qualifications was raised by Senator Anderson. If this portion of the testimony is to be included in the published proceedings, it would be desirable, for the sake of completeness, that a full record of my qualifications be included as well. The attached curriculum vitae is somewhat more complete than the record to which Senator Anderson referred and which I believe was the Men of Science abstract.

I am also attaching a list of my publications. I do not wish that this list be included in the proceedings but I simply submit it as a matter of record for the files of the subcommittee.

I continue to hear favorable reports about the hearings. We all look forward to the final proceedings.

With best regards.

Sincerely,

MERRIL EISENBUD, *Manager.*

BIOGRAPHICAL DATA, MERRIL EISENBUD, JUNE 10, 1957

Date of birth: March 18, 1915, New York City
Education: New York University, College of Engineering (1932-36) B. S. in E. E. 1936
Positions held:
- Industrial hygienist, Liberty Mutual Insurance Co., 1936-47
- Chief, Industrial Hygiene Branch, Health and Safety Laboratory, United States Atomic Energy Commission, 1947-49
- Director, Health and Safety Laboratory, United States Atomic Energy Commission, 1949 to present
- Manager, New York Operations Office, United States Atomic Energy Commission, 1954 to present
- Senior scientific advisor, Preparatory Commission of the International Atomic Energy Agency, 1957

Lecturer, Columbia, School of Public Health, 1945-50
Adjunct associate professor, department of sanitary engineering, New York University, 1945-50
Associate professor, industrial medicine, New York University, department of industrial medicine, 1950-55
Adjunct professor, industrial medicine, Postgraduate School of Medicine, New York University, 1955-
Committees:
- National Research Council:
  - Toxicology Committee, 1952-
  - Committee on Atmospheric and Industrial Hygiene, 1952-
- American Standards Association:
  - Subcommittee on Radium, Dust, and Radon Gas, Z37, 1949-
  - Sectional Committee on the Use of X-Rays, Z-54, 1951-
- National Safety Council: Executive committee, Chemical Section, 1951-
- Radiological Advisory Committee: Office of Civil Defense, City of New York, 1950-
- Technical advisor, United States delegation, U. N. Conference on the Peaceful Uses of Atomic Energy, Geneva, 1955
- Alternate United States representative, U. N., Scientific Committee on the Effects of Atomic Radiation, 1956-.

Member, National Academy of Sciences Committee on the Meteorological Aspects of the Effecvts of Atomic Radiation 1956–.
Aspects of the Effects of Atomic Radiation, 1956–.
Scientific adviser, United States delegation, Conference on the Statute of the International Atomic Energy Agency, 1956–.
Member, World Health Organization, Expert Advisory Panel on Radiation, 1957–.

Memberships:

American Industrial Hygiene Association (board of directors, 1955–58), American Public Health Association, New York Academy of Science, Radiation Research Society, American Association for the Advancement of Science.

LIST OF PUBLISHED WORK OF MERRIL EISENBUD

Global Distribution of Strontium-90 from Nuclear Detonations, Scientific Monthly (May 1957), pp. 237–244.
Monitoring Network for Measuring Radioactive Fallout, J. Am. Water Works Association, Vol. 48, No. 6 (June 1956).
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Industrial Hygiene of Uranium Processing, A. M. A. Arch. Ind. Health, Vol. 14, pp. 12–22 (July 1956) (with J. A. Quigley).
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Radioactive Fallout in the United States, Science, Vol. 121, No. 3150, pp. 677–680 (May 13, 1955) (with John H. Harley).
Health Hazards from Beryllium, Chapter 12, published in The Metal Beryllium by the American Society for Metals, 1955.
Industrial Hygiene and Medical Survey of a Thorium Refinery, Arch. of Industrial Health (March 1955) Vol. 11, pp. 234–242 (with Roy Albert, Paul Klevin, John Harley, J. Fresco and W. B. Harris).
How Important Is Surface Contamination?, Nucleonics (August 1954) Vol. 12, No. 8, pp. 12–15 (with Hanson Blatz and Eugene V. Barry).
Radioactive Dust From Nuclear Detonations, Science (Feb. 1953) Vol. 117, No. 3033, pp. 141–147 (with John H. Harley).
Dust Sampler Which Simulates Upper and Lower Lung Deposition, Arch. Ind. Hyg. and Occup. Med. (November 1953), Vol. 8, pp. 446–452 (with W. B. Harris).
Field Equipment for the Collection and Evaluation of Toxic and Radioactive Contaminants, Arch. Ind. Hyg. and Occup. Med. (June 1953) Vol. 7, pp. 490–502 (with W. B. Harris and H. D. LeVine).
Retention, Distribution and Elimination of Inhaled Particulates, with Particular Reference to the Evaluation of Radiologic Risk, Arch. Ind. Hyg. and Occup. Med., (September 1952) Vol. 6, pp. 214–225.
Epidemiology of Beryllium Intoxication, Arch. Ind. Hyg. and Occup. Med. (August 1951) Vol. 4, pp. 123–151 (with James H. Sterner).
A Method of Obtaining Reproducible Breath Radon Samples, Arch. Ind. Hyg. and Occup. Med. (July 1951), Vol. 4, pp. 1–9 (with John H. Harley and Evelyn Jetter).
Meterological Technics in Air Pollution Surveys, Arch. Ind. Hyg. and Occup. Med. (January 1951), Vol. 3, pp. 90–97 (with W. B. Harris).
Radiation Hazards in the Atomic Energy Program, Ind. Med. and Surgery, (January 1951), Vol. 20: 1, 7–11.
Fire Protection Precautions for Uranium Scrap and Powder, Nucleonics (May 1950), Vol. 6, No. 5, pp. 34–37 (with Edward J. Kehoe and Francis L. Brannigan).
Nonoccupational Berylliosis, J. Ind. Hyg. and Toxicol. (1949) Vol. 31: 5 (with R. C. Wanta, Cyril Dustan, L. T. Steadman, W. B. Harris and B. S. Wolf).
Environmental Studies In Plants and Laboratories Using Beryllium: The Acute Disease, J. Ind. Hyg. and Toxicol. (1948) Vol. 30: 5 (with C. F. Berghout and L. T. Steadman).
Health Hazards In Aircraft Manufacturing, Industrial Medicine (March 1949) Vol. 18: 3, pp. 99–102.
The Comparative Performance of Air-Supplied Welding Helmets, Welding Journal (April 1948) (with Leslie Silverman).

Mercury Exposures In Dry Battery Manufacture, J. Ind. Hyg. and Toxicol. (November 1947) Vol. 29, No. 6 (with Charles R. Williams and Stanley E. Pihl).
Control of the Lead Poisoning Hazard in Can Manufacturing, J. Ind. Hyg. and Toxicol. (December 1945), Vol. 27, No. 10 (with J. W. McEwan).
The Principal Health Hazards in Metal Finishing Departments and Their Control, Metal Finishing (1942).

Mr. EISENBUD. Thank you.

I am deeply gratified, Mr. Chairman, at the opportunity to correct the record with respect to the item which Dr. Lapp has included on page 2, paragraph C, in which he quotes a New York newspaper sentence and illustrates what, I think, needs not be illustrated; namely, the danger of taking something out of context. The date of that quotation is March 20, 1955. This was a very jittery period. This was immediately following the announcement within the AEC of the results of the March 1, 1954 detonation in the Pacific. It also coincided with the beginning of the Teapot series of detonations in Nevada, which, if my recollection is correct, began a week or two prior to this announcement, or perhaps shortly thereafter.

In any case, the fact that the first post-Castle detonations were about to take place in Nevada was very much in the minds of many of our citizens.

This reporter came to me to find out whether, in my opinion, the kind of accident which occurred in Bikini in 1954 could happen in this country as a result of the test being contemplated or already underway in Nevada. We were not talking about the long-range hazards of fallout. We were not talking about strontium 90. We were talking about the kinds of acute effects which one had, unfortunately, seen in both the Marshallese and Japanese fishermen in 1954.

My comment, which may or may not have been quoted accurately—I really don't know—had to do, primarily, with the relationship of acute effects to the kinds of radiation levels that are expected from the Nevada test, which are of the order of 1 milliroentgen or thereabouts, at least, in the United States.

Senator ANDERSON. Mr. Eisenbud, this statement was made on March 20, 1955. I understand you do not question the accuracy of the quotation.

Mr. EISENBUD. I do not question the accuracy of the quotation nor will I certify it nor will I certify the accuracy of Dr. Lapp's quotation of a quotation.

Senator ANDERSON. If it should prove he is accurate you are willing to stand by it.

Mr. EISENBUD. I am willing to accept this version of it.

Senator ANDERSON. The testimony introduced by Dr. Lapp and the chart introduced showed that we had the Castle series in 1954 in which we had put in the atmosphere more fission products perhaps in that year than we have put in all the rest of the tests together. Therefore we were at our highest peak when you made your comment. The figure was several times above the figure which Dr. Langham has said is a safe figure year by year.

It is your testimony that we could do a million times that and do no damage.

Mr. EISENBUD. No, sir. This was not the question which was asked of me. I was asked specifically whether there was any possibility in the eastern United States of an accident which would produce the

kinds of illness among the people that were seen in the Marshallese and Japanese fishermen.

This would require around 100 roentgen. The radiation doses which had been observed were something less than a milliroentgen. This is a ratio of about a million to one. When you put it into context it is perfectly accurate.

Senator ANDERSON. That is what I am trying to get to. We had just finished the Castle series. You would agree with that; would you not?

Mr. EISENBUD. That was a year before.

Senator ANDERSON. We had finished them in 1954 and this statement was made March 20, 1955. We had finished the Castle series, had we not?

Mr. EISENBUD. Yes, sir.

Senator ANDERSON. You said the total fallout to date from all tests would have to be multiplied by a million to produce visible deleterious effects except in areas close to the explosion itself. It is your testimony that having put that year into the stratosphere, or whatever fallout pattern there was, something in the neighborhood of 30 megatons of fissionable products, that we would have to put a million times that for it to have any ill effect except in the immediate vicinity of the test. Is that correct?

Mr. EISENBUD. That is incorrect.

Senator ANDERSON. You think that is a somewhat reckless statement?

Mr. EISENBUD. This is not what I said.

Senator ANDERSON. That is what I am trying to get to. The paper misquoted you.

Mr. EISENBUD. No, sir. I have not seen that quotation in 2 years. This sentence is out of context.

Senator ANDERSON. I am reading it to you. If it should prove to be an exact quotation, is it a reckless statement?

Mr. EISENBUD. Out of context; no.

Senator ANDERSON. In or out of context, is it a reckless statement to say that the Castle test which included an extremely large shot which turned loose in one series of tests as much fission products as probably all the rest of the tests by all the rest of the countries? If you can place reliance on the data gathered at Los Alamos, do I understand in context or out of context, you could have a million times that and have no visible deleterious effects except in the immediate area? Do you wonder that looking at that statement Dr. Lapp thought it might be a slight degree of recklessness?

Mr. EISENBUD. Sir, I would like Dr. Lapp to comment on what I have——

Senator ANDERSON. The point is that they wanted to give you an opportunity, and this is your day.

Mr. EISENBUD. Yes, sir. I think Dr. Lapp will probably understand what I am talking about. Let me say this, sir: I think it is a great misfortune——

Senator ANDERSON. Dr. Lapp was treated critically because he had used this language. They said in all fairness you would have an opportunity to reply. This is your hour for fairness. Why don't you go ahead and reply?

Representative COLE. I think he would if you let him.

Mr. EISENBUD. I think it is a great misfortune, sir, that to add to public confusion we find it necessary to discuss in the same session the long-range hazard and the close in hazard. For most of these sessions we have been talking about the long-range hazard from strontium 90 and from the gamma radiation.

In this interview I was talking about the close-range hazard, the kind of hazard that may develop within a few hours after detonation and the kind of hazard which produced illness in the Marshallese and Japanese in 1954.

Senator ANDERSON. Then from the close-range hazard—we will just confine it to that—since we put some 30 megatons of fission products in the atmosphere in the Castle test just passed, you believe that we could safely put a million times that in a single year in a test without doing any damage?

Mr. EISENBUD. Certainly not, sir.

Senator ANDERSON. What were you talking about? If it is not from the close range or long range, what other range is there?

Mr. EISENBUD. I was talking about the immediate gamma radiation from the fallout which occurs in the eastern United States within a matter of a day or so after a detonation in Nevada. This is not in the statement because the statement has been taken out of context.

Senator ANDERSON. Could that have been apparent if Dr. Lapp had read the whole interview?

Mr. EISENBUD. I can't vouch for the validity of the interview, sir. I do not recall this.

Dr. LAPP. I would like to make this statement in all fairness to Dr. Eisenbud, and I certainly do not mean to attack his integrity. The reason for quoting these and the use of the word "reckless" was to demonstrate the need for being quite exact when dealing with such a touchy subject as radioactivity. I am trying to interpret this recklessness not in terms of Dr. Eisenbud's personal recklessness but in terms of how it may appear to people who read these things and who do read single sentences.

Senator ANDERSON. I quite agree with you. I tried to say earlier that I think Mr. Eisenbud is a very fine public servant. I went up to him and told him that I appreciated very much the testimony he gave the other day. I did not regard your statement about him as a vicious attack upon him. But if it comes down to whether it was reckless, there are people who read that statement alone who would think it had just a slight tinge of recklessness in it, since the Castle test had just been finished.

Dr. LAPP. May I ask a question of Dr. Eisenbud? Is this permitted?

Representative COLE. Don't look at me.

Representative HOLIFIELD. I think——

Dr. LAPP. I would like to ask one single question.

Representative HOLIFIELD. You might ask the Chair a question.

Dr. LAPP. I would like to ask the Chair a question, as to what the roentgen dosage on the fallout on Troy, N. Y., was.

Representative HOLIFIELD. The Chair has been told, but the Chair has temporarily forgotten now. The Chair will ask Dr. Eisenbud if he recalls.

Mr. EISENBUD. Yes, sir. It has been variously estimated.

Mr. EISENBUD. It is unclassified. It was published 3 years ago. The upper limit of estimate is something under 100 milliroentgen. It loses about 1 milliroentgen. I would personally estimate it at about 10 milliroentgen.

Representative HOLIFIELD. The Chair thanks the gentleman for that information.

Representative COLE. Could I inquire, in order for the information to be helpful, if these samplings that occurred were related to what tests? When were the samplings taken which you say indicated an upper limit of 100 milliroentgen?

Mr. EISENBUD. I believe it was the third or fourth test of the series held in Nevada in the spring of 1953. There was a rain out over the Troy-Albany area which coincided with the passage of a cloud from Nevada. So that a very large percentage of this cloud was washed down.

Senator ANDERSON (presiding). Are there additional questions or statements?

Representative VAN ZANDT. Dr. Eisenbud, I understand your position is that the statement that Dr. Lapp attributes as being a reckless one was taken out of context.

Mr. EISENBUD. Yes, sir.

Dr. LAPP. Is it proper for me to respond? I have done a little arithmetic. Let us take 10 milliroentgens, as Dr. Eisenbud estimates, and we multiply 10 milliroentgens. That would be .01 roentgens by 10 to the sixth, which will give us 10 to the fourth, which is 10,000 roentgens.

Senator ANDERSON. 10,000 roentgens would kill everybody in sight.

Mr. EISENBUD. Yes.

Senator ANDERSON. So that would mean there would not be any immediate danger if you kill everybody in sight?

Representative PRICE. Mr. Chairman, one of the points that we do not want to overlook is that Dr. Lapp is trying to point out the responsibility of the Commission to release information as promptly as possible so that these types of statements would not be made. Is that not one of the reasons for you citing this statement?

Dr. LAPP. I really feel if we had better relations here between the press and the Atomic Energy Commission we could in a minute avoid much of this difficulty.

Representative PRICE. That is the reason I understood that you gave this example.

Dr. LAPP. Yes.

Senator ANDERSON. May I say, Mr. Eisenbud, that I am truly sorry that Dr. Lapp's quotation has caused any embarrassment. I want to repeat what I said before. I certainly regard you as a fine public servant doing a good job. I am very happy that you are here make your contribtuion today.

Mr. EISENBUD. Thank you, sir.

(The full statement of Dr. Ralph E. Lapp follows:)

STATEMENT OF RALPH E. LAPP ON RADIOACTIVE FALLOUT

EXPLANATORY NOTE

Mr. Holifield, I received your invitation to testify before this committee while I was in Japan. I cut short my trip in order to attend the hearings. May I

say that I appreciate very much this opportunity to appear here. I would like to add that I am very gratified that your investigations to date have thrown so much light on the problem of radioactive fallout. I believe that these hearings will stand as a landmark in the history of our knowledge about this relatively new phenomenon.

COMMENT ON DR. LIBBY

Appearing as I do after Dr. Libby, I would like to comment on his contributions to fallout. Dr. Libby has not only stimulated extensive research in fallout investigations such as Project Sunshine, but he has also taken the initiative in publication of his findings. I feel very strongly that he deserves a great deal of credit for his work on fallout. Were it not for Dr. Libby we might well be confronted with a considerably smaller body of knowledge about fallout than we have today.

MY INTEREST IN FALLOUT

I have had an active interest in atomic bomb phenomenology ever since I witnessed the Bikini Baker test in the summer of 1946. However, my interest in radioactive fallout was really stimulated by the 1954 Bravo test at Bikini. This was the test which resulted in radioactive contamination of the *Lucky Dragon No. 5*, a Japanese tuna trawler.

My initial interest in fallout centered upon civil defense. In this connection, I published a series of articles on fallout in the Bulletin of Atomic Scientists as follows:

November 1954: Civil Defense Faces New Peril
February 1955: Radioactive Fallout
June 1955: Radioactive Fallout III
November 1955: Global Fallout
September 1956: The "Humanitarian" H-Bomb
October 1956: Strontium Limits in Peace and War.

NATURE OF MY TESTIMONY

I am dividing my testimony into four parts:

I: General Remarks
II: Local Fallout
III: Remote Fallout
IV: Constructive Proposals

Because of the number and complexity of the topics covered, I am presenting my remarks in terse or fragmentary form. This will permit the committee to bypass topics of less importance and concentrate upon those of more concern.

PART I. GENERAL REMARKS

A. Necessity for numbers

Public confusion about fallout will continue to increase unless scientists can provide a quantitative or semiquantitive evaluation of the various hazards associated with fallout. Precision is probably not possible due to the nature of the hazards and we may have to be content with numbers which vary by a factor of 2, 3, or even 10. Th is committee has already performed a valuable service in narrowing the range of estimates made by individual witnesses.

B. Disagreement among scientists

The public is apt to conclude that if scientists cannot agree upon the hazard, then all is confusion. It would be nice if the scientists could all agree upon a quantitative estimate of the hazard, which could then be given to the public. Two unusual circumstances have combined to produce the current confusion on fallout.

First, the urgency of our times has focused attention upon problems for which science did not have textbook answers. Available knowledge was inadequate and research had to be initiated to provide answers.

Second, the ordinary process by which scientists argue out their answers was interdicted by the complexity of the problem and secrecy. Scientists outside the Atomic Energy Commission have full-time jobs and could scarcely be expected to tunnel into the complexities of the problem in a few leisure hours.

C. Responsibility of the Atomic Energy Commission

Considering these factors, I think that the AEC has the responsibility for providing the outside world with the facts about fallout as promptly as these become available. Scientists, technicians, and officials of the AEC must present only reasoned and careful estimates of the hazards based upon factual knowledge. Reckless or nonsubstantiated statements do a disservice to the AEC and to the Nation.

Example: Dr. Eisenbud is quoted in an article titled "Man Who Measures A-Fallout Belittles Danger" (Sunday News, New York, March 20, 1955) as follows: "The total fallout to date from all tests would have to be multiplied by a million to produce visible, deleterious effects except in areas close to the explosion, itself."

Example: Dr. Libby in a speech dated June 3, 1955, stated: "However, as far as immediate or somatic damage to the health is concerned, the fallout dosage rate as of January 1 of this year in the United States could be increased 15,000 times without hazard."

Example: Dr. Richard Doan while in Tokyo on May 13, 1957 stated that the bomb tests would not have "the slightest possible effect" on humans.

I do not label Dr. Libby's statement as reckless but interpose it to illustrate the spectrum of opinion being given to the public.

D. World interest in fallout

I am informed by a cable from Tokyo that the deliberations of this committee hearing are being "splashed across page 1" of the Japanese newspapers. This comes as no surprise to me for my trip through Japan alerted me to the overwhelming interest manifested there in atomic radiation.

The committee might be interested in my observation that fallout has become an acute weapon for propaganda. For example, I found that the Japanese scientists are actively studying the radioactivity of their tea because of the assertion from the Chinese mainland that Japanese tea is radioactive. Some people in Japan are so keenly aware of fallout that they take showers after being out in a rain. The great public outcry against the British Christmas Island tests, but there was no great demonstration against Soviet tests. It is a great victory for psychological warfare experts when they can induce selective sensitivity to fallout.

America puts itself in a bad light when it fails to present its case clearly to the world. Even casual analysis of the news reporting in this country will show that AEC pronouncements on fallout are not received with full credibility. This situation is obviously not in the full interests of national security.

E. The nature of biological data

It is inherent in the very nature of the biological research into the effects of radiation upon humans that a high degree of accuracy is not attainable, especially on a human experience basis. As Dr. Langham of the Los Alamos Laboratory has testified human experience with retention of radium 226 is the basis for setting upon a maximum permissible concentration (MPC) for radiostrontium (Sr–90). Yet our actual experience is confined to a small sample of acutely exposed individuals and a small sample of less acutely exposed people.

Actually, our concern should focus not upon acute effects in man which are highly unlikely from peacetime bomb testing, but rather with the chronic, debilitating long-term effects from irradiation of humans. We must be conscious of the need to appraise long-delayed effects, say, 50 years after entry of radioelements into the body. Here our knowledge is quite limited.

F. Radiation limits for a global population

I would like to stress the fact that consideration of safe limits for irradiation of the world's population is essentially a new problem. Prior to the awareness of global fallout, the International Commission on Radiological Protection made its recommendations for those who would be exposed to radiation in pursuit of their occupation. Such groups initially were numbered in the hundreds and then in the thousands as atomic energy came of age. Individuals within such groups were healthy adults exposed to known and restricted hazards; they were subject to administrative controls and medical supervision.

In setting up limits for a total population, we must take into account the varying radiosensitivity of individuals, the complete spectrum of age, the persistence of the hazards, the lack of medical control, the varying degrees of health of people and the variety of their diet. Yet it was not until last year that the

Atomic Energy Commission introduced the difference between an occupational MPC and a global MPC into its releases on fallout.

In view of the nature of our knowledge and the totality of the sample with which we are dealing, I would urge a big factor of safety in setting limits to bomb testing. It would be tragic to find some day that we had erred in setting the limits.

G. Soviet nuclear tests

On my recent trip to Japan, I learned that Japanese scientists collected sufficiently active samples from Russian tests to perform radiochemistry upon the bomb debris. I am informed that five Soviet tests produced a fallout on Japan from which scientists measured and identified the presence of uranium 237 (U-237). Soviet explosions characterized by such fallout were judged to be in the megaton range. These estimates are subject to considerable uncertainty but one authority told me that he estimated at least two bomb yields in the range of 10 megatons.

Two Soviet nuclear tests were observed to originate in the arctic region whereas the remaining tests took place in a region estimated to be Ozero Balkash (Lake Balkhash) which is southeast of the new coal area of Karaganda. The air mass trajectories from central Siberia frequently sweep across the islands of Japan, especially Hokkaido. They also produce tropospheric fallout over the United States as well. Here in Washington you could swipe a Kleenex over a car top and cause a Geiger counter to respond readily.

The presence of U-237 in the Soviet fallouts proves that the Soviets have achieved a compound fission-fusion or multiple-stage weapon. According to my information, this was first accomplished in September 1954.

I would like to add that I am informed by Japanese sources that Soviet tests produce 70 percent of the fallout observed on Japan. Pacific tests account for 20 percent and the Nevada shots add 10 percent.

PART II. LOCAL FALLOUT

Definition: By local fallout, I mean that which comes to earth within several hundred miles of the explosion site and is deposited within the first day or so. The following points are discussed with relation to the direct effects of external radiation. My interest centers upon the problem of civil defense in dealing with the radiation hazard in a contaminated area.

A. Areas of contamination

It is evident from the analyses such as Dr. Schafer presented to this committee that a nuclear attack upon the United States would involve an overlapping or smeared out pattern of bomb fallouts, especially over Northeastern United States. In making assessment of the radioactive power of bomb fallout, it is useful to introduce a new unit "the eternity roentgen square mile." This is a measure of the irradiating power of bomb fallout. By "eternity roentgen" I mean the total roentgens accumulated in dosage from 1 hour to eternity. This unit is then multiplied by square miles over which fallout occurs.

Example: To estimate the eternity roentgen square mile contamination from a 15 megaton explosion we proceed as follows. Assume that the ratio of fission to fusion energy release is 2:1. Then 10 megatons of fission energy will be involved. Assume a 50 percent local fallout. This yields 5 megatons of fission products in the fallout area. Simple calculation shows that 1 megaton of fission products could contaminate (if uniformly deposited at 1 hour) 1,000 square miles so that the 1 hour to infinity dose in open air would be 6,000 roentgens. Thus 5 mt. of fission products could raise this dosage to 30,000 r. Or if the area of fallout were greater, say, 5,000 square miles, the dosage would be 6,000 r.

An attack such as Dr. Schafer assumed involved 2,500 mt. of bomb yield and he specified "dirty weapons" surface burst. By "dirty" it is meant that the ratio of fission to fusion is fairly high. If we assume 2,000 mt. of fp (fission products) locally deposited this amounts to a total of 12 billion roentgen square miles of potential contamination. Obviously, this is a maximum since much fallout will occur after 1 hour; this will dissipate harmlessly in the air until it is deposited on the ground. Nontheless, the figure especially for surface burst bombs where local contamination will be maximized gives an indication of the magnitude of the fallout hazard. If any considerable fraction of this total figure is concentrated on a relatively small area, such as Northeastern United States industrial heartland, the corresponding radiation intensities will be severe.

Example: If a 1,000 mt. of fp concentrated upon Northeastern United States much of the region would be subject to a fallout of about 10,000 eternity roentgens. This would correspond to a fallout intensity of 2,000 r./hr. at 1 hr. I shall discuss the significance of such fallout in section C.

B. Clean and dirty bombs

The above discussion should make it obvious that the fallout from dirty weapons is of immense importance because of the area contaminable with a medium-weight attack. However, it may be useful to compare the damage areas of the two types of weapons.

A clean or relatively clean air burst bomb would have to depend upon blast and heat for its destructive effects. Consider, for example, the areas hit by the blast of a 20 mt. bomb.

Blast overpressure (pounds per square inch)	Distance in miles	Area in square miles
100	1.5	7
10	7.5	175
3	15.0	1,600

For purposes of comparison, one might select purely military targets such as air fields, missile sites and "hardened" targets which would require up to 100 pounds per square inch blast overpressure for destruction. Under such cases the aiming accuracy in delivery would have to be very great if you wished to "hit". A miss by as little as 2 miles with a clean bomb could be considered a complete miss. If one is concerned with population bombing and the criterion is the destruction of a framehouse, the 3 pounds per square inch blast would be appropriate. A greater aiming error would be allowable but one would not want to miss by more than 10 miles.

To complete the comparison, it is necessary to assess the persistence of the radioactive effect of fallout to discover whether such contamination would be effective in denying land to normal or to even emergency use.

I make this point, not to assert that there would be no military uses for clean bombs, but to emphasize that from the standpoint of civil defense, it might be very misleading to assume that an enemy would forego the use of dirty bombs.

C. Persistence of fallout

Witnesses before this committee have testified as to the rapid decay of fission products in fallout. It is true that a fresh mixture of bomb-produced fission products exhibits rapid decay. Half of the radioactivity, as measured from a time base at 1 hour, disappears in 32 hours. The AEC states in its report The Effects of High-Yield Nuclear Explosions (February 1955) "The main radioactivity of fallout decreases very rapidly with time—for the most part, within the first hours after the explosion."

Section 10.1 of the "Effects of Nuclear Weapons" (June 1957) states: "The radiation intensity decreases rapidly with time and except for areas of very high initial contamination, it ceases to be a serious hazard within a few weeks."

These statements, it seems to me, give the impression that civil defense has nothing to worry about after a few days, or a few weeks. I believe that the discussion in section A (part II) coupled with Dr. Schafer's estimates of the fallout intensities show that large areas of the United States could be contaminated to the extent of 2,000 roentgens per hour at 1 hour. The following schedule of roentgen dosages results:

From 1 hour[1] through end of 1st day	4,700 r.
From end of 1st day to end of 1st week	1,730 r.
From end of 1st week to end of 1st month	920 r.
From end of 1st month to end of 1st year	1,060 r.
From end of 1st year to 50 years[2]	840 r.

[1] The dosage of 4,700 r. depends upon fallout at 1 hour after detonation. Fallout at later times (i. e., farther downwind) would significantly reduce this first day dose.

[2] Terrain and weathering would play a significant role in reducing this dose.

If we look only at the decay rate, the decay seems rapid. Starting at 1 hour after the explosion, the rate would be 2,000 r./hr. At 7 hrs. it would decrease to 200 r./hr., at 1 day to 45 r./hr. At the end of 2 days it would be 20 r./hr. At

1 week it would be 4.2 r./hr. or 100 roentgens per day. At the end of 1 month it would be 17 r./day; this would decrease to 4 r./day at 100 days, and to 0.8 r./day at 1 year.

D. Significance of local fallout

I believe that the combination of the vast areas contaminable with high-yield thermonuclear weapons with the long term persistence of the fission products poses a problem for civil defense of great magnitude as different from that of the A-bomb as that was from the TNT bomb.

E. Genetic consequences of a 2,500 mt. attack upon United States

Assume, as in section II–A, that the United States is hit by a 2,500 megaton attack, in which some 2,000 mt. of fission products are deposited on the ground. As we have seen this can be expressed as a contamination equivalent to 12 billion roentgen square miles, where the roentgen as used here is the eternity roentgen. If we make the simplifying assumption that this contamination is spread uniformly over the continental United States, this will produce an eternity exposure of 4,000 roentgens (land area is 3 million square miles). In an actual situation this would be unevenly distributed but the probability is that exposures would be greatest nearest inhabited areas, so this calculation probably underestimates the effect.

Let us assume that people go into hiding and receive no significient radiation exposure in the first month after the attack. I grant that this is highly unlikely so the calculation is again underestimated. From 1 month to 30 years the exposure would average 20 percent of 4,000 roentgens or 800 r. Divide this in half to take account of weathering, so that we get 400 r. as the average exposure to every American who survives. We may now apply Dr. Crow's data of June 4 to this figure of 400 r. Instead of 2 billion children in the next generation we consider one-twentieth this figure. This means that we multiply Dr. Crow's values by 400 and divide by 0.1 (Dr. Crow's assumed 30-year exposure) times 20. Thus we multiply all his figures by a factor of 200. This yields:

Effect	1st generation	Total
a. Physical and mental defects	1,600,000	16,000,000
b. Stillbirths and childhood deaths	4,000,000	120,000,000
c. Embryonic and neonatal deaths	8,000,000	140,000,000
d. Intangible defects	(1)	----------

[1] A larger but unknown number.

In the first generation about 2 out of every 10 children would be genetically defective. The sum total of all deferred deaths from the attack would be 272 million or several times the number killed by the direct attack. In addition almost everyone would shoulder an increased genetic burden.

PART III. REMOTE FALLOUT

NOTE.—I believe that previous testimony and the roundtable discussions on the production, injection, transport, and fallout of radioactive debris from bomb explosions provide a more solid base for evaluating the hazard. It is obvious that in the area of the uptake of fission products there still remain some unknowns which future research and global survey will resolve. It is doubtful if the uncertainties inherent in estimating the biological effects of radiation will be resolved as readily. I wish to comment specifically upon several topics which I feel deserve amplification and emphasis.

A. The "present test rate"

This term has been used frequently before this committee as well as in the public domain during the past few years. Rarely has it been defined in a quantitative manner. To interpret the meaning of the present rate of testing one has to specify a precise number of megatons of fission products injected into the stratosphere.

I do not profess to have any inside information upon which to base an estimate of the present rate of testing. However, it is instructive to consider how the test rate has progressed since 1945. I am drawing upon data openly available and I apologize for the roughness of the data. Nonetheless, the progression of the annual increments to the curve may be worth considering.

The curve I shall plot covers the period from 1945 to 1957 with a projection to the future. Extrapolation of the curve will be considered. First, we plot the total fission yield of all bombs tested in each year. Up until 1952, and more generally until 1954, this total fission yield will correspond roughly to the total bomb yield. After that time corrections have been estimated for the fission fraction of the explosive yield. Second, we then estimate the fraction of the fission debris which is injected into the stratospheric reservoir and is globally dispersed. This fraction will depend upon bomb yield and firing conditions.

I claim no accuracy for the estimate and present the curve for qualitative illustration of the trend in the bomb test rate.

As long as weapons tests were confined to pure fission weapons of relatively low yield the global fallout of strontium 90 would be negligible. With higher yield fission weapons, more of the fission products began to be injected into the stratosphere and retained there for global distribution. However, because of the economy limits (cost of fissionable material) the global hazard was still quite small.

On November 1, 1952, the United States entered a new domain of weapons testing. The Soviet Union followed suit within a year. These tests were then followed by the Castle series of tests in the Pacific in the spring of 1954 when high-yield contaminating bombs were tested at Bikini. These tests added to the stratospheric reservoir the majority of the radiostrontium still present there. Dr. Langham of the Los Alamos Laboratory testified before this committee that one might assume an average rate of 10 megatons of fission products per year. This total of 50 Mt. for the past 5 years would check with the data that I have estimated.

In order to define the present test rate one has to wait until the end of the year, add up the Soviet, United States and United Kingdom contributions to the stratosphere and thus reach a reasonable figure. In the absence of international exchange of data, this is done through remote instrumentation.

B. A limit to bomb tests

Dr. Neuman in testifying before this committee arrived at a "safe" test rate of 2.2 megatons of stratospheric strontium 90 per year; i. e., the amount associated with the annual injection of strontium 90 produced in an explosion such that 2.2 megatons of fission energy inject their fission products into the stratosphere. Some time ago, I estimated on a similar line of reasoning that this "safe" limit would correspond to 3 megatons (plus or minus a factor of 3 times this value); i. e., as high as 9 or as low as 1 megaton per year. I believe this is in general agreement with the 2 to 10 megatons estimated by your round-table discussion of May 29.

Superimposing these values upon the chart for the yearly test rates, it is seen that this "safe" limit was exceeded in 1954, 1956, and probably will be exceeded this year as well.

I believe the concept of a safe testing rate is of very great importance from a global health viewpoint. But the limit seems so low that it would appear that setting up a quota for each nation's annual testing would de doomed to the same fate as the attempt to control battleship construction in the 1920's.

But I believe that an internationally constituted monitoring system could keep systematic check on the level of fission products which fall out as a result of bomb tests. The publication of these measurements would have a profound effect on world opinion.

It would be of interest to learn from the Atomic Energy Commission the value for the safe annual limit which it assumed in its deliberations during the past 3 years.

C. A limit to war

There is obviously a great difference between the risks which a nation and its people take in time of war. During wartime the "safe" limit for the military application of thermonuclear weapons would be, at least in my opinion, 50 times greater than the peacetime safe limit. Whatever the value, the concept of a limit to the use of nuclear weapons in war is quite new. I have in mind here the inevitable lashback which an aggressor would suffer from the fallout of his own bombs. Dr. Libby, in his speech of January 19, 1956, estimated that between 330,000 and 440,000 megatons of bombs would have to be exploded before "the likelihood of untoward effects would be appreciable."

In view of the consequences to humanity, I wonder whether the Atomic Energy Commission or the Department of Defense has ever prepared a full study of the biological consequences of a nuclear war.

D. Evaluation of risk

Dr. Eisenbud's remark that he is not troubled by the hazard of giving milk containing traces of Sr-90 to his children illustrates, I believe, an extreme form in which a radiation risk may be assayed. The probability of injurious effect to a sample of three people is very small; from a personal viewpoint it is probably negligible. But if one applies the same probability of injury to 3 billion people, then even a very minute effect becomes significant to those whose lives are affected. Suppose we deal with a probability of one in a million. For a global population this would involve about 3,000 people. Such an effect would, from a personal viewpoint, constitute a very small risk. The same line of argument applies to Dr. Shields Warren's reference to the personal danger from the radiation emitted by the microgram of radium in his luminous wristwatch dial. The fact that he observes no visual change in the skin directly beneath the wristwatch proves nothing—certainly, it does not constitute a test of a radiation threshold.

If I may comment on the testimony of other expert witnesses who testified on Monday, June 3, I find Dr. H. L. Friedell's philosophy with regard to bomb tests rather whimsical. Apparently, Dr. Friedell believes we do not have enough data to evaluate the risk and we should proceed on a path of blissful optimism.

Testimony introduced on behalf of Dr. Jacob Furth by Dr. Shields Warren contains a recommendation that "the burden of decision rests not with biomedical investigations, but with military experts." In a matter so rooted in nuclear science and so veiled in conflicting opinions, I am reluctant to entrust the burden of the decision to military experts.

Dr. E. P. Cronkite cites three reports as authoritative in evaluation of strontium risks: (*a*) the United Nations report, (*b*) the British Medical Council report of June 1956, and (*c*) the National Academy of Sciences report of June 1956.

(*a*) So far as I know the United Nations report on strontium has not been concluded. I am informed it will not be published this year.

(*b*) With regard to the British report, I would call attention to the last sentence of that report. "Nevertheless, if the concentration in human bones showed signs of rising greatly beyond one-hundredth of that corresponding to the maximum permissible occupational level it would indicate the need for immediate consideration of the problem."

(*c*) With regard to the National Academy study, may I refer to page 60 of the general report, section 3: "However, if the testing programs of the several countries producing theromonuclear weapons were to intensify, strotospheric storage time may become a critical item in terms of the hazard to mankind."

Since the National Academy report was issued the United States, the U. S. S. R., and the United Kingdom have all tested theromonuclear weapons. I submit that the testing programs are intensifying.

E. Future nuclear tests

In assessing the future commitments of radioactive debris to the earth's atmosphere, we must deal with many unknowns. Had we attempted an estimate 5 years ago, prior to Castle-type weapons, we would have arrived at most misleading and optimistic projections. The end of weapon development is not in sight and no one can say that unexpected developments may not occur.

For example, may not smaller nations be stimulated by British success with thermonuclear type weapons and place maximum emphasis upon such development?

Additionally, can we be sure that a nation would restrain itself and not test a 100 megaton dirty weapon if military requirements and nuclear technology indicated that such a weapon was desirable?

Will the requirements of adapting maximum megatonnage to a small warhead put emphasis on further development of dirty type weapons?

We cannot answer these questions at this time, but we do know that a single weapons test of very high fission yield can add a strontium burden to the atmosphere far beyond the limits we have been discussing.

The United States has contributed the largest fraction of radiostrontium to the stratosphere and I think that it is most encouraging that the fullest discussion of the strontium fallout should occur in this country. I am not aware of any large body of published information on this subject of Soviet origin. It is known, however, that the Soviets are engaged in strontium studies.

In concluding this section, I would like again to stress Dr. Libby's contributions to this subject. They are of very great value and I feel sure that we would be in a much poorer position today to evaluate the strontium problem were it

not for Dr. Libby's personal interest in this field of investigation and the research which he has promoted so vigorously.

F. The strontium problem

It is clear from testimony given to this committee that data on the fallout of radiostrontium are becoming more firm as research results come in. I think that scientists can agree on the pattern of strontium fallout around the world. We are in a poorer position in our knowledge of strontium uptake in the biosphere. I am disturbed by fluctuations which have occurred in AEC statements on discrimination factors in the uptake of strontium into the food chain. Discrimination factors are high in the milk link of the food chain, but are much lower in foods consumed directly by humans. A discrimination factor of 7 is estimated by Dr. N. S. MacDonald of UCLA and Dr. W. Neuman estimates an average discrimination factor of 8.

Strontium 90 determinations in man must be expanded to assess the increase in strontium 90 burden with time. Careful determinations of natural strontium in humans deserve increased attention. We know that more strontium 90 will accumulate in humans as a result of bombs tested in the past and as a result of current tests. The determination as to how much of this radioelement may be tolerated safely is a matter for the biologists to discuss. This committee has heard a fairly wide range of opinion from its expert witnesses on the probable biological effects of Sr-90 levels in man. But it seems to me that even in this area some agreement was reached, especially when Dr. Shields Warren stated on June 3:

"I would be reluctant to see the average strontium 90 content of bones, particularly in children, go much above 10 times the present level."

Dr. Libby's speeches show that Sr-90 fallout will continue and the strontium 90 level in human bones will increase.

Unless restraints are imposed upon commitments of fission products to the atmosphere, it is only a matter of time before the strontium 90 level of Dr. Warren is reached.

PART IV. CONSTRUCTIVE PROPOSALS

A. Atomic Energy Commission information policy

I suggest that this committee or its parent committee may wish to review the information policy of the AEC with regard to nuclear weapon effects, with a view toward revising this policy so that information may be made available more promptly and completely. I believe that the national interest demands a much better relation between the press and the Atomic Energy Commission.

B. Report on the probable biological consequences of a nuclear war

I suggest that the Joint Committee on Atomic Energy might wish to recommend or sponsor the preparation of an analysis of the probable biological effect of nuclear warfare. It would be useful to investigate probable lashback effects from various levels of nuclear bombardment.

C. Data useful to civil defense

I believe that the committee's investigations have produced information of critical value to civil defense planning. It might be useful to have a summary report of these data transmitted to the Federal Civil Defense Administration. I have not seen many representatives of the Federal Civil Defense Administration at these hearings.

D. Research in long-range estimation of nuclear explosives

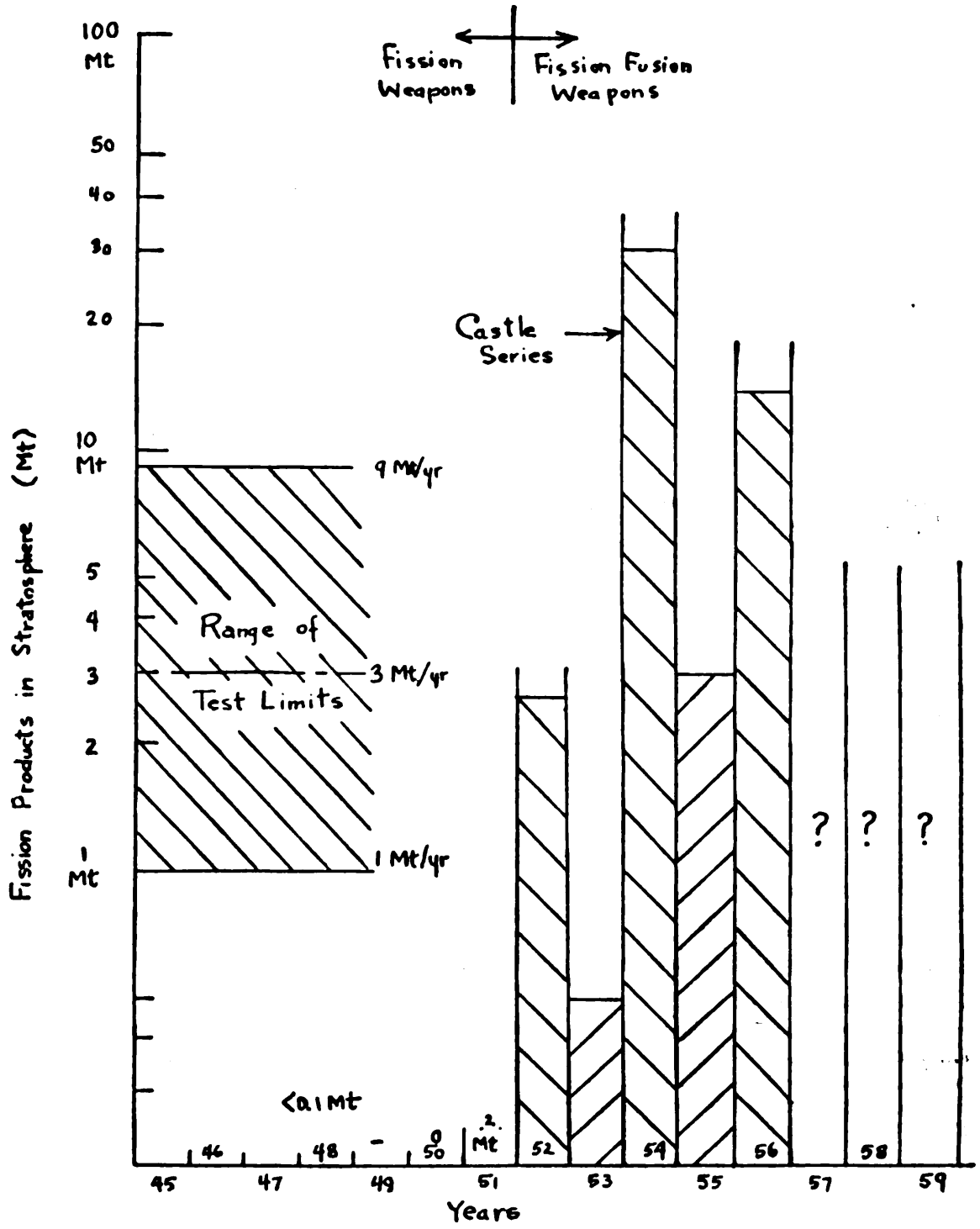
It is known that considerable effort has focused on long-range detection of nuclear detonations. Attention should be given to the declassification of such data as would bear upon evaluation of the radiostrontium problem. Other data would be most useful in discussion of the feasibility of policing an agreed upon test limit.

E. An annual fallout report

In view of the great public concern over fallout hazards, I would urge that the Atomic Energy Commission be required to issue an annual report on the degree of fallout and its uptake in biological systems. Perhaps such a report should be prepared by a university task force.

F. National radiation control

I would urge that the Congress continue its investigations of radiation hazards, extending them into the broader area of peacetime uses of radiation. I believe that the ever-increasing uses of radiation must be subject to legislative controls. Radiation protection in the United States needs uniform legal status.



Rough Estimate of Annual Additions to Stratosphere From Nuclear Weapons Tests of U.S.A And U.S.S.R [in Megatons of Fission Products]

The maximum permissible level of Sr^{90} for workers is set at 1 μc in the total adult skeleton, and the recommended level for the general population is set at 0.1 μc[4] (9, 32, 33). The permissible levels of radiation exposure (including that from Sr^{90}) is predicated on the assumption that chronic and/or delayed effects of radiation are threshold phenomena (Fig. 8). That is to say, there is a threshold dose below which effect rapidly becomes insignificant and above which effect increases expenentially over a limited dose range. If this is indeed the case, 100 $\mu\mu c$ Sr^{90}/g Ca must be looked upon as a true maximum permissible level and not an average value for the general population.

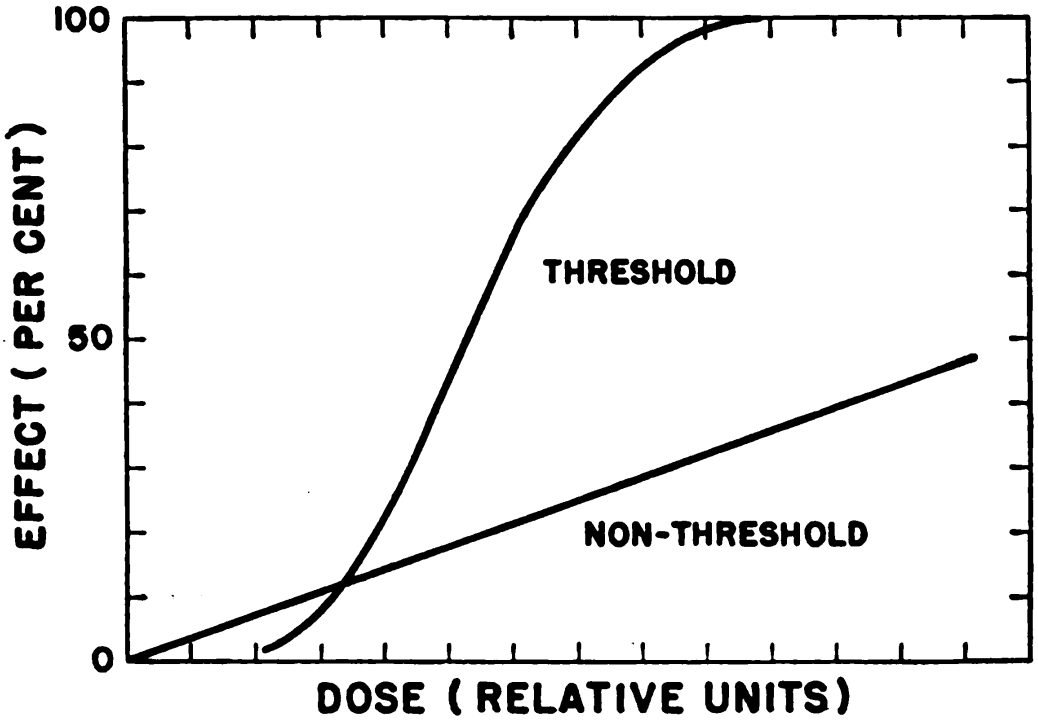


Figure 8.—Threshold versus nonthreshold effect in relation to increasing radiation dose.

Threshold versus Nonthreshold Response.—At the present time it is impossible to say whether leukemogenic and sarcogenic responses to chronic radiation dosage are threshold or nonthreshold relationships. Argument for a linear relationship between incidence of leukemia and radiation dose was presented recently by Lewis (34). His argument was based on all major sources of human data and included a consideration of the Japanese atomic bomb survivors, the British cases of X-ray treated spondylitis patients, X-ray treated cases of thymic enlargement, practicing radiologists and spontaneous incidence of leukemia in Brooklyn, New York. Radiation as a carcinogenic agent has been discussed at length by Brues (35), who stated that the relation between radiation dose and carcinogenic effect is not easy to find and a critical experiment has yet to be done which will clearly indicate, even in a single instance, what the relation is over more than a small range of dosages. While admitting that it is not known, he proposes that a threshold relationship between radiation dose and tumor incidence does exist (36).

Genetic response to external radiation indeed appears to be linear and a given increment of dose produces a corresponding equal increment of effect, regardless of position on the dosage scale (Fig. 8). If it is assumed that all chronic effects of radiation are linear, it would seem more reasonable to establish permissible levels for the general population on the basis of probability of risk averaged over the entire group. Present incidence of bone sarcoma and of leukemia averaged over the entire population is about 2 and 6 per 100,000, respectively. About 10 percent of the natural incidence of leukemia (34) (and perhaps of bone sarcoma) may be attributable to natural radiation background. If this is true, doubling the natural background dose to the bone might be expected to increase the inci-

[4] There is about 1 kg of calcium in the adult human skeleton; therefore, the MPL of Sr^{90} in the general population is equivalent to 0.1 μc Sr^{90}/kg Ca=100 $m\mu c$/kg Ca=100 $\mu\mu c$/g Ca=100 Sunshine Units (2).

dences of bone tumors and leukemia to 2.2 and 6.6 per 100,000, respectively. Such a small increase distributed through the general population may be undetectable, and 100$\mu\mu$c Sr^{90}/g Ca (which would about double the background skeletal dose) may be regarded by some as an acceptable average maximum equilibrium for the general population.

B. Hazard from Present and Predicted Sr^{90} Levels

The significance of the general hazard of present and predicted levels of Sr^{90} in bone can be evaluated only in relation to human experience, which is indeed inadequate. Bone sarcoma has resulted from a fixed skeletal burden of 3.6 μc of pure Ra^{226}, and nondeleterious bone changes have been observed in persons having only 0.4 μc for a period of 25 years (37). Necrosis and tumors of the bone have occurred also several years after large doses of X-ray (38), and consideration of human experience with leukemogenic effects of X and gamma radiation (9, 34, 39) suggests that about 80 rads may double the incidence of leukemia.

The only other human experience with which present and predicted levels of Sr^{90} may be compared is that arising from natural background radiation. Natural background dose to the bone (during a 70-year lifetime) may vary from about 8 to 38 rem (40). The major contribution to background variation is differences in the radium levels of soils and minerals. The average natural skeletal radiation dose rate was carefully evaluated by Dudley and Evans (41) and their data are shown in Table VII.

TABLE VII.—*Average natural background radiation dose rate to the skeleton (Dudley, Evans)*

Source of radiation	Skeletal dose rate (mrem/year)	Total dose to age 70 (rem)
K^{40} (internal)	8	0.56
Ra^{226} (internal)	12	0.84
MsTh (internal)	12	0.84
RaD (internal)	12	0.84
Cosmic rays (external)	30	2.1
Local gamma rays (external)	60	4.2
Total	134	9.4

Table VIII (after Brues (42)) gives a general summary of estimated skeletal radiation doses from accepted maximum permissible levels and from present and predicted Sr^{90} burdens in relation to human experience. The maximum permissible level of Sr^{90} (100 $\mu\mu$c/g Ca) is estimated to deliver about 8 rads[a] to the skeleton during a 70-year life-time. This is comparable to the average natural backgroumd dose to the bone for the same time period and a factor of ~4 below the maximum natural background dose to which small segments of the general population may be exposed as a result of differences in altitude and natural radium content of soils and minerals. It is a factor of 40 below the lowest skeletal dose which has produced minimal nondeleterious bone changes. These data suggest that the present average maximum Sr^{90} equilibrium level in children will result in a life-time radiation dose of approximately 2 per cent of the accepted maximum permissible level for the general population. The predicted average maximum level of Sr^{90} (from bone data) in about 1975, assuming no further weapons tests, corresponds to a skeletal radiation dose of about 2.6 per cent of the maximum permissible level with a spread ($\pm 3\sigma$) of about 0.5 to 6 per cent.

The biological significance of present and predicted Sr^{90} average maximum equilibrium levels and maximum permissible levels for occupational and non-occupational exposure is summarized in Table IX.

[a] Eight rads is the calculated dose assuming incorporation to age 20 and decay to age 70 with no more incorporation. If equilibrium were maintained, the calculated skeletal dose would be about 21 rads. Since some but not all of the skeleton undergoes remodeling plus exchange, somewhere between 8 and 21 rads is probably more correct.

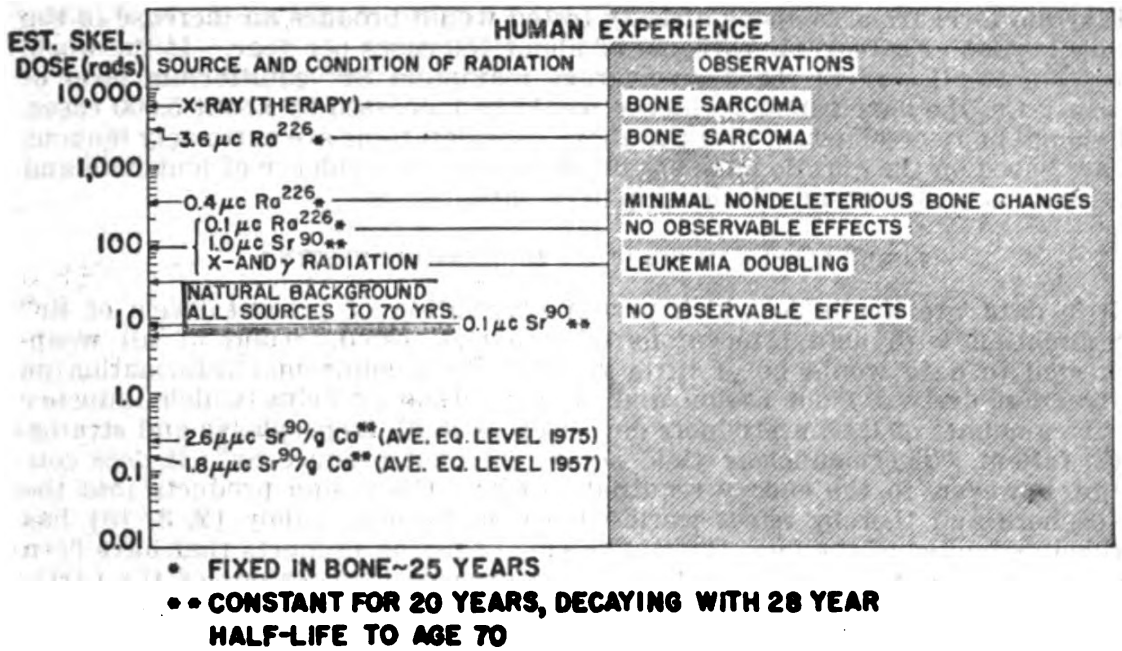


TABLE IX.—*Biological significance of present and predicted Sr^{90} average maximum equilibrium levels and maximum permissible levels for occupational and nonoccupational exposure*

Sr^{90} level	MPL nonoccupational exposure (100 μμ c/g)	MPL occupational exposure (1,000 μμ c/g)	Minimum bone changes	Minimum sarcoma dose	Leukemia doubling dose
Present (1.8 μμ c/g Ca)[1]	1/50	1/500	1/2,000	1/20,000	1/500
Predicted (2.6 μμ c/g Ca)[1]	1/40	1/400	1/1,400	1/14,000	1/360
100 μμ c/g (MPL nonoccupational exposure)	----------	1/10	1/10	1/100	1/10
1,000 μμ c/g (MPL occupational exposure)	10	----------	1	1/10	1

[1] Average maximum equilibrium level of Sr^{90}, probability slight that many will run more than 3 times average.

The most interesting comparison made in Table IX is that between Sr^{90} levels and the leukemia doubling dose, assuming a nonthreshold relation between incidence and radiation exposure. These data indicate that the predicted average maximum equilibrium level of Sr^{90}, assuming no more weapons tests after Operation Redwing (Fall 1956), is 1/360 of the leukemia doubling dose. Theoretically, this level is equivalent to an increase in leukemia incidence of 1.7 cases per 10 million population. The rate in some localized areas may be several times higher, but averaged over the world population of 2.6 billion, this would produce an increased lukemia burden of about 400 cases per year. If the entire world population is allowed to reach an average maximum Sr^{90} equilibrium level of 100 μμc/g Ca, the average increase in world's leukemia burden would be about 16,000 cases per year, or about 5 to 10 per cent (34).

The above discussion entails the assumption that Sr^{90} beta radiation induces leukemia of the bone marrow origin at the same rate (per unit of absorbed dose) as X and gamma rays. Much of the beta radiation from Sr^{90} will be absorbed in the bone and not reach the hematopoietic tissues at all. Experiments by Brues *et al* (43) suggest that Sr^{90} (half-life 55 days, $E\beta$=1.5 Mev.) administered to mice is relatively more spectacular as an osteosarcogenic agent than a leukemogenic agent. Furthermore, leukemia was not a significant finding in the radium dial painters (29, 44) or in the radium-injection cases (37).

Human data on radiation-induced osteogenic sarcoma are not adequate to provide a basis for a sarcoma doubling dose or for an estimation of the per cent of normal incidence attributable to natural background. If, however, the same assumptions used for leukemia are applied to osteogenic sarcoma (nonthreshold response, 10 per cent of normal incidence of 2 per 100,000 attributable to natural background and a doubling dose of 80 rads), the predicted average maximum Sr^{90}

equilibrium level from weapons already tested would produce an increase in the world's burden of osteogenic sarcoma of about 150 cases per year. If the world population is allowed to reach an average maximum Sr^{90} equilibrium level of 100 $\mu\mu c/g$ Ca, the bone tumor incidence would be increased by about 5,000 cases.

It should be re-emphasized that the above considerations are extremely tenuous and are based on the questionable assumption that the incidence of leukemia and bona sarcoma bear a linear relationship to radiation dose.

7. SR^{90} LEVELS IN RELATION TO WEAPONS TESTING

Little data are available which permit correlation of present levels of Sr^{90} contamination with actual megatons of weapons tested. Total of all weapons tested to date would be of little value without additional information on the fraction derived from fission and on conditions of firing which influence relative amounts of fission products deposited as local, tropospheric and stratospheric fallout. Thermonuclear yield *per se* does not produce Sr^{90}; it does contribute, however, to the energy required to carry the fission products into the stratosphere and thereby effect world-wide distribution. Libby (2, 3, 10) has provided estimates of the megaton equivalents of fission products that have been injected into the stratosphere and deposited over various regions of the earth. Based on these values, Kulp (24) estimated that present levels (Fall 1956) of environmental contamination (including the stratospheric reservoir) was the result of injection of products from about 50 MT of fission yield.

By simple proportionality he estimated biospheric injection of Sr^{90} from 35,000 MT of fission would bring the average maximum Sr^{90} equilibrium bone level of the world's population up to 100 $\mu\mu c/g$ Ca (the MPL for occupational exposure). He used 1.3 $\mu\mu c$ Sr^{90}/g CA as the average maximum equilibrium level for the world population in 1970, assuming no more weapons tests. Kulp did not say that the average maximum level of the world population should be allowed to reach 1000 $\mu\mu c$ Sr^{90}/g Ca,[6] but apparently tried to show the relation between megatons of fission tested to date and a MPL familiar to all (45). Libby (2, 8) also has used the occupational MPL as a reference point in discussing the hazard to the world population. This practice has led to confusion of the public and criticism of the Atomic Energy Commission (46). The data in Table X (based on various estimates of 1970 equilibrium bone levels) show the estimated megaton equivalents fission yield that may be injected into the biosphere (all at once) to bring the Sr^{90} average maximum equilibrium bone values in the United States, the northern hemispheric fallout belt and the world up to the limits set for occupational and nonoccupational exposure. The table also shows the influence of various factors for nonuniformity of distribution and uptake on the estimates for the northern hemispheric fallout belt (in which the majority of the world's population is distributed) based on the average maximum level derived from bone data. *These data are presented primarily to emphasize the principal areas of uncertainty responsible for apparent disagreements among various authorities.*

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[6] The Sr^{90} maximum permissible level accepted by the National and International Commissions on Radiological Protection as being applicable to large segments of the population is equivalent to 100 $\mu\mu c/g$ Ca.

TABLE X.—*Sr^{90} levels in relation to megaton equivalents of fission products injected into the biosphere*

Source of equilibrium estimate and area	Estimated equilibrium 1970–75 ($\mu\mu c$ Sr^{90}/g Ca)	MT required to produce average MPL	
		(1,000 $\mu\mu c$/g Ca)	(100 $\mu\mu c$/g Ca)
United States (average):			
Libby (10), Ecological data	1.7–3.9	30,000–13,000	3,000–1,300
Kulp (24), Ecological data	2	25,000	2,500
This report, Ecological data	3.1	16,000	1,600
This report, Bone data	3.2	16,000	1,600
Eisenbud (4) [1], Milk data	4.1	12,000	1,200
This report, Milk data	3.5	14,000	1,400
Area 10° N. to 60° N. latitude (average):			
This report, Ecological data	2.5	20,000	2,000
This report, Bone data	2.6	20,000	2,000
World (average):			
Kulp (24), Ecological data	1.3	38,000	3,800
This report, Bone data	1.7	30,000	3,000
This report, Ecological data	1.6	30,000	3,000
Ave. bone data (10° N. to 60° N. latitude)	2.6	20,000	2,000
Ave. × ½ (for nonuniformity (10))		4,000	400
Ave. × ⅒ (for nonuniformity) [2]		2,000	200

[1] Eisenbud's value corrected for discrimination factor of 0.5.
[2] Indicated by spread in current bone data from all ages (24).

Inspection of the data in Table X shows a variation of about 300 in the megaton equivalents of fission products that may be injected into the biosphere, depending on whether one wishes to be ultraconservative and use the highest equilibrium bone value, the nonoccupational MPL and the largest safety factor for nonuniformity, or use the occupational MPL applied to the world average maximum bone level with no safety factor for nonuniformity. The most important point to these data is that they show that the major portion of the variaion is associated with two factors, (1) the maximum permissible level for Sr^{90} as applied to the fallout problem, and (2) the factor for nonuniformity of Sr^{90} distribution and uptake.

The most important question regarding the potential hazard of world-wide fallout to the general population is its relation to future weapons testing. If there is an upper limit to the amount of Sr^{90} that can be tolerated in the bones of the population, then the number of megaton equivalents of fission products that can be contributed per year to the biosphere by all nations must be limited.

Theoretically, the total yearly injection rate should be that amount which, at equilibrium, will not result in a significant fraction of the population exceeding the limit of safety. If a constant yearly injection rate of 1, 10 or 100 MT of fission is adhered to, in about 100 years the amount of Sr^{90} added to the environment will come into equilibrium with the rate of Sr^{90} decay, and continuation of weapons testing at that rate will result in no further increase in the average maximum equilibrium level in the bones of the population. At that time the average equilibrium bone level will be directly proportional to the yearly injection rate, i. e., if 10 or 100 megaton equivalents are injected per year, the average equilibrium bone level will be 10 and 100 times higher, respectively, than it will be if only 1 megaton equivalent is injected.

Only three unclassified reports concerning implications of future biospheric fission product injection rates have appeared. Campbell (5) mathematically related surface deposition to a constant stratospheric injection rate. His equation suggests Sr^{90} surface deposition levels may reach 30 times present values with continuation of the present rate of biospheric contamination for 100 years. His approach, however, makes no allowance for the tropospheric deposition rate. Libby (49) has developed an expression relating Sr^{90} surface deposition to a constant test rate, using a calculated ratio of rates of stratospheric to tropospheric injection of 4 to 7. Libby's calculation is based on a measured value of 30 mc Sr^{90}/mi^2 for the present cumulated fallout in the northeastern United States (one of the more highly contaminated spots in the world) and the assumption that the stratospheric reservoir presently contains the Sr^{90} from 24 MT of fision. Libby first estimated that the maximum equilibrium Sr^{90} deposition level should approach 8 times the present level (10). On re-

calculation of the ratio he obtained a value of 11 (49).[7] Using a build-up factor of 8 and assuming an ecological discrimination factor of 20 to 80 against Sr^{90} in going from soil to human bone, he estimated an average maximum bone equilibrium level of 5 to 20 $\mu\mu c$ Sr^{90}/g Ca for the population of the United States when equilibrium is reached. If the present rate is continued for 28 years, the average maximum level would reach only one-half of the above values (10). The general consensus of opinion (23) of the various specialists in the field of bone and mineral metabolism is that the discrimination factors used above are too high and that a factor of 10 to 20 is more realistic.

Stewart, Crooks and Fisher (48) estimated the concentration of Sr^{90} on the ground in the United Kingdom as 4.5 mc/km^2 on January 1, 1956, and the mean deposition rate as 2.3 mc/km^2/year. From these data the ground concentration in the U. K. as of January 1, 1957, would be about 17 mc/mi^2. They also estimated that an equilibrium value of about 500 mc/mi^2 (200 mc/km^2) of Sr^{90} may be reached in the U. K. in about 100 years if the present rate of biospheric contamination is continued. These data show a build-up factor of about 30 over present levels.

Predicted average maximum Sr^{90} bone levels, assuming continuation of the present biospheric contamination rate for about 100 years, are given in Table XI. These values were calculated from present (Fall 1956) bone equilibrium levels derived either from analyses or from ecological considerations assuming an ecological discrimination factor of 10 against Sr^{90} and Libby's (49) build-up factor of 11 for continued testing. The one British value mentioned above is given for comparison.

TABLE XI.—*Average maximum Sr^{90} bone equilibrium levels assuming continuation of past 5-year injection rate for 100 years*

Basis of estimate and area	Average maximum equilibrium bone level	
	Fall 1956 ($\mu\mu c$/g Ca)	In 100 year [1] ($\mu\mu c$/g Ca)
United States:		
Libby (10), ecological data	1.7–3.9	[2] 19–43
Kulp (24), ecological data	1.4	15
This report, ecological data	3.6	40
This report, bone data	2.5	28
This report, milk data	2.5	28
Eisenbud, milk data	4.1	45
Area 60° N. to 10° N. latitude:		
This report, ecological data	2.6	29
This report, bone data	1.8	20
Stewart, et al. (48), present soil data United Kingdom [3]	3.1	34
Stewart, et al. (48), United Kingdom soil in 100 years	----------	[4] 90
World average:		
This report, ecological data	1.3	14
This report, bone data	.9	10
Kulp (24), ecological data (1970)	1.3	14

[1] Based on, or 5MT of fission yield per year assuming testing for infinite time, an equilibrium level, with continued testing, of 11 times the present level.
[2] Libby's estimate (10) was 5 to 20 $\mu\mu c$/g Ca.
[3] Assuming an ecological discrimination factor of 10 against Sr^{90}.
[4] This value corresponds to a buildup factor of about 30.

The discrepancy between the British value and the others is immediately apparent. No details as to the basis of their estimate were given but it is possible that no consideration was given to the ratio of rates of stratospheric and tropospheric injection, since it compares favorably with the value that would be predicted from the derivations made by Campbell (5).

The data in Table XI (excluding the British value) suggest that, continued biospheric contamination at the present rate for 100 years might result in average maximum equilibrium bone levels of about 30, 25 and 12 $\mu\mu c$ Sr^{90}/g Ca for the United States, the area between 60°N–10°N latitude and the world, respectively.[8]

[7] R. K. Zeigler of LASL Theoretical Division has confirmed the calculation using Libby's assumptions.
[8] If Machta's concept of uneven stratospheric fallout is indeed the case, the average Sr 90 values for the United States and the area between 60° N–10° N latitude may be increased by about a factor of 2.

The upper limit that might be expected in the United States, assuming a factor of 5 as adequate to make allowances for non-homogeneities of Sr^{90} deposition and uptake, would approach about 150$\mu\mu$c Sr^{90}/g Ca, or 150 percent of the accepted maximum permissible level. After 30 years of testing, average maximum equilibrium bone levels may approach one-half of the above values, which may result in an upper limit for the United States of about 75 percent of the maximum allowable level. Assuming the average yearly injection of fission products during the past five years equal to about 10MT of fission yield, it would seem that the testing of 10MT of fission per year by all nations for 30 years should probably be considered the upper limit, or 5MT of fission yield per year assuming testing for infinite time. If these are the limits of acceptable injection rate, international agreement not to exceed these levels seems desirable. Present levels of Sr^{90} contamination are due almost entirely to tests held by only two nations. Present and predicted future Sr^{90} levels, even if weapons tests are continued at the present rate for a few years, does not seem dangerous. However, indiscriminate testing of high-fission yield weapons by many nations could result in serious levels of worldwide contamination.

SUMMARY

What does the accompanying mass of technical data mean with regard to the controversy over cessation or continuation of nuclear weapons tests? Nowhere in this report has a recommendation been made either to stop or to continue testing. Such a recommendation requires a careful weighing of the importance of the nation's nuclear weapons capability in averting a nuclear war, against the probability that a few people might get leukemia or bone sarcoma or manifest a genetic abnormality who otherwise might not have done so. Therefore, the decision to stop or continue tests requires a value judgment involving knowledge of the potential seriousness of present and future threats to the national security, and whether they should or should no be stopped on the basis of moral and humanitarian principles is not readily amenable to solution by the scientific method.

The purpose of this report is to evaluate, as factually as possible from existing data, the potential hazard of Sr^{90} fallout. Evaluation of existing data supports the following general conclusions:

(1) Radioactive isotopes deposited in the bone in sufficient quantity will produce serious consequences, including bone cancer and leukemia. Present Sr^{90} levels in the bones of the population are quite low. The present average maximum equilibrium Sr^{90} radiation dose to the bones of young children is greater than that for adults and is about 2 per cent of the average dose received from unavoidable natural background radiation contributed by cosmic rays and by radium, thorium, uranium, etc., in the environment. It is about 2 per cent of the maximum permissible level adopted by the National and International Commissions on Radiological Protection as acceptable for large segments of the general population. The present Sr^{90} radiation dose to adults, averaged over the total skeleton, is about one-tenth of that to children. Because of nonuniformity of fallout and individual variations in uptake and deposition of Sr^{90} in bone, a very small number of people may accumulate a skeletal dose that will be about five times the average, and an equal number will accumulate only about one-fifth the average. Since in the stratosphere there is still some Sr^{90} from past weapons tests, the average radiation dose may continue to rise until about 1975 even if no more weapons tests are held. At that time the equilibrium level may be 3 to 4 per cent of the average natural background.

(2) If Sr^{90} contamination from weapons testing by all nations continues at the same rate as has occurred during the past five years (about 10 megaton equivalents of TNT fission yield per year), equilibrium will be reached in about 100 years. At equilibrium the amount of Sr^{90} which will disappear each year from our environment due to radioactive decay will just about equal the amount that is being produced each year. At this time continuing weapons tests will not result in any further increase in Sr^{90} in the bones of the population. Assuming a buildup factor of 11 for equilibrium levels, the average Sr^{90} radiation dose to the bones of the population of the United States is predicted to be about 30 percent of the average radiation dose from natural background, or about 30 percent of the maximum permissible level adopted by the National and International Commissions. Since a factor of 5 may be necessary to allow for nonuniformities in fallout and bone uptake, the Sr^{90} radiation dose to a few individuals may approach 150 per cent of the recommended maximum level as an upper limit. If strato-

spheric fallout is not as uniform as predicted by the Libby model, the above values may be somewhat higher. Thirty years of testing will result in an average Sr^{90} level of about one-half of the equilibrium value, which may result in a few people approaching 75 percent of the recommended maximum permissible radiation dose. On this basis, limitation of biospheric contamination by all nations to about 10 megaton equivalents of fission yield per year for 30 years, or 5 megaton equivalents per year, indefinitely, might be desirable.

(3) The existing data support the conclusion that the present rate of biospheric Sr^{90} contamination, if continued for 20 to 30 years, will not produce average maximum population bone levels that will exceed the maximum permissible levels accepted by the National and International Commissions on Radiological Protection. The data also show that many nations cannot test high fission yield weapons indiscriminately and indefinitely without running the risk of seriously exceeding these recommended levels. For this reason, international agreement to limit testing might be desirable while negotiating for agreement to stop testing altogether.

(4) The data presently available definitely show that the greatest question concerning world-wide Sr^{90} contamination concerns the decision as to the *ACCEPTABLE* maximum permissible body dose for the general population in terms of the individual and in terms of world health. The answer to this question involves moral and humanitarian principles, as well as scientific uncertainties as to the biological consequences.

Some deductions as to the worst biological consequences of Sr^{90} fallout (which excludes the genetics question) can be made by accepting two rather pessimistic assumptions, neither of which has been proved, but both of which seem conservative. The natural yearly incidence of leukemia in the United States is about six cases per 100,000 population, and the incidence of bone tumors is about two cases per 100,000. Therefore, normally there are about 10,000 cases of leukemia in the United States per year and about 3,000 cases of bone tumors. This was the natural incidence even before any radioactive fallout had occurred; so atomic bombs have had nothing to do with it. The first assumption is that any amount of radiation has a small chance of producing tumors and leukemia. That is, the assumption is made that there is no *absolutely safe* radiation dose and any amount is theoretically bad. This assumption is open to serious question and is one of the major points of disagreement among scientists. If all radiation is bad, the natural background radiation which we cannot avoid must be responsible for a fraction of the six leukemia cases and the two bone cancer cases per 100,000 that occur in the population. The second assumption is that about 10 percent of the normal incidence of bone tumors and leukemia is due to natural background radiation. There is some scientific evidence from X and gamma radiation in support of this assumption as applied to incidence of leukemia, but none for Sr^{90} radiation. There is no evidence to support its application to bone cancer. We do know, however, that there are things in our environment other than radiation which will cause bone cancer; so it certainly would not be right to attribute 100 per cent of the bone cancer incidence to natural background.

On the basis of the assumptions given above, the incidence of leukemia in the United States might increase from 10,000 cases per year to about 10,030 and the incidence of bone cancer might increase from about 3,000 to about 3,010. This suggests a total increase of 40 cases out of a population of 165 million people as the maximum biological consequence of Sr^{90} fallout from weapons tested to date *by all nations*. If the present rate of biospheric Sr^{90} contamination continues for 30 years, the biological consequence of Sr^{90} fallout may be a total increase in the United States population of about 250 cases per year of these two diseases. There is a good chance that this prediction may overestimate the population risk. The assumption that even the smallest amount of Sr^{90} deposited in bone carries a small probability of harm is seriously questioned, and there is a possibility that a threshold below which no leukemia and bone cancer will be produced does exist. Animal experiments with radioactive strontium definitely indicate that Sr^{90} may not be as bad as anticipated for producing leukemia because the beta radiation from the Sr^{90} gets absorbed in the hard bone and does not radiate the bone marrow where leukemia begins. These same experiments do suggest that production of bone tumors by Sr^{90} is about as expected on the basis of human experience with radium.

(5) The present data also indicate that much more research on both the physical and biological factors of fallout must be done if weapons tests are to be continued. This research is necessary to narrow the limits of error and uncertainty in existing data and to permit predictions to be based on facts instead of on what appears to be reasonable assumptions. More research on the biological and

medical effects of radiation and radioactive materials is essential, even to the future of the power reactor program which also produces Sr^{90} and other fission products.

Although present knowledge of the biological effects of radiation and radioactive materials is not all it should be to allow plunging ahead recklessly and without worry into all aspects of nuclear technology, it is adequate to dispel an attitude of gloom and doom. Radiation is not the only potential hazard man is facing as part of the price of living in a highly developed society nor is it the insurmountable one.

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The committee will stand adjourned.

(Whereupon, at 12:15 p. m., Thursday, June 6, 1957, the committee adjourned, to reconvene at 10 a. m., tomorrow, Friday, June 7, 1957, in the old Supreme Court chamber, the Capitol.)

Leukemia in exposed persons—Number and rate by presence of radiation symptoms and distance from hypocenter

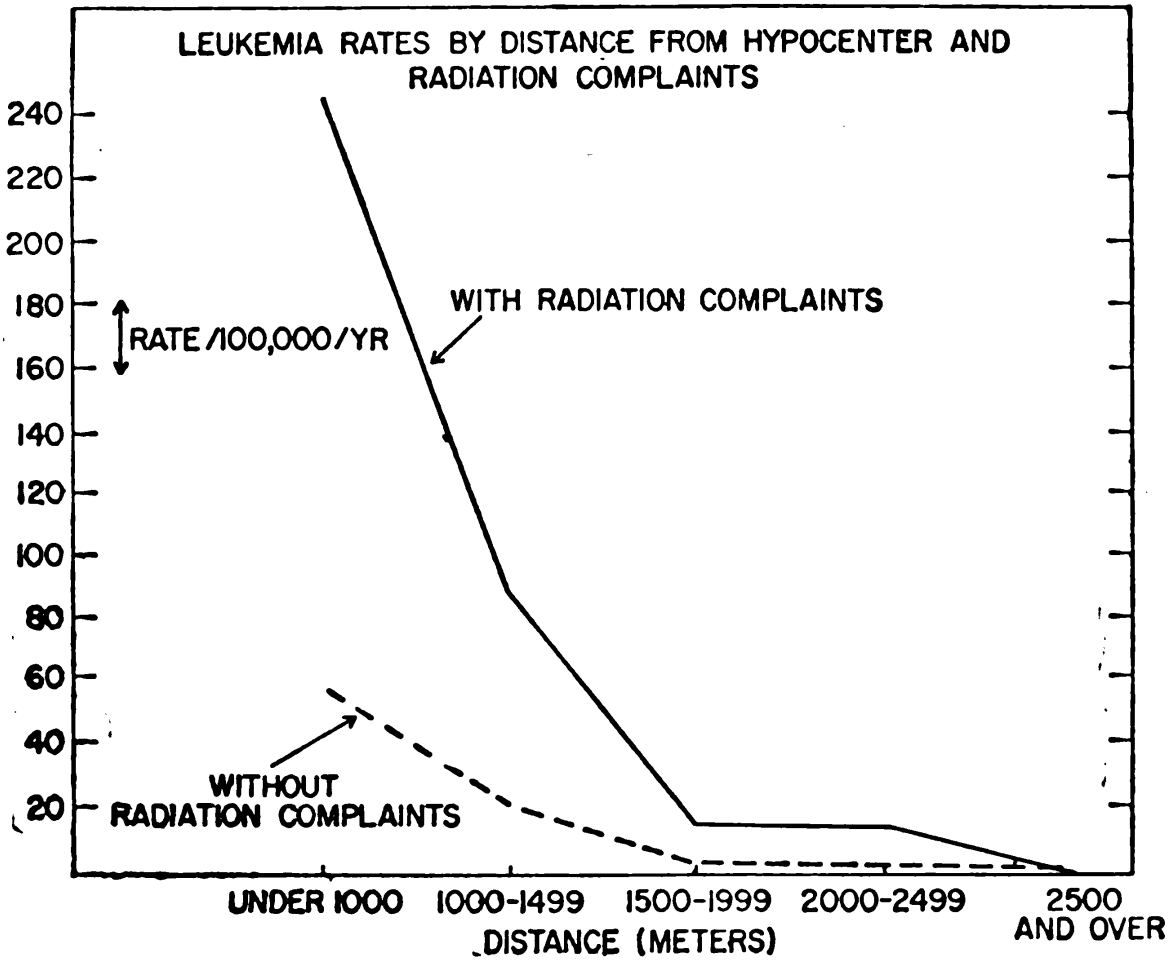
Distance from hypocenter—meters	Hiroshima population [1]			Number of cases of leukemia [2]			Incidence		
	SRC [3]	NRC [4]	Total	SRC	NRC	Total	SRC	NRC	Total
Under 1,000	750	450	1,200	14	2	16	246.2	58.6	175.8
1,000 to 1,499	2,250	8,250	10,500	15	13	28	87.9	20.8	35.2
1,500 to 1,999	1,750	16,950	18,700	2	4	6	15.1	3.1	4.2
2,000 to 2,499	950	16,250	17,200	1	1	2	13.9	0.8	1.5
2,500 over	850	49,650	50,500	0	8	8	--------	2.1	2.1
Total	6,550	91,550	98,100	32	28	60	64.4	4.0	8.1

[1] Source: Population estimated and rounded off to the nearest 50 persons. These population figures were based on the Commission's 1949 Radiation census and the Japanese national census (1950). Numbers of survivors with severe radiation complaints were estimated from observations made by the Commission's genetics department on 19,675 Hiroshima survivors of childbearing age.

[2] Source: Listing of Leukemia Cases in Hiroshima and Nagasaki, Sept. 1955. Cases are restricted to those in persons resident in Hiroshima at the time of diagnosis, and described in the listing under the heading, Diagnosis Acceptable.

[3] SRC: Significant radiation complaints—Epilation or purpura on history not confirmed by competent physical examination or medical records.

[4] NRC: No radiation complaints.



Leukemia in persons exposed within 1,500 meters of the hypocenter—Number and rate, by sex and age ATB [1]

Age ATB	Hiroshima population, 1950		Number of cases of leukemia [2]		Incidence, annual rate per 100,000	
	Male	Female	Male	Female	Male	Female
0 to 9	839	878	6	6	94.3	90.1
10 to 19	995	1,490	7	2	92.8	17.7
20 to 29	458	1,352	3	6	86.4	58.5
30 to 39	713	1,118	3	2	55.5	23.6
40 to 49	902	1,016	3	2	43.9	26.0
50 to 59	606	572	1	2	21.8	46.1
60 to 69	236	278	----------	1	----------	47.4
Total	4,749	6,704	23	21	63.8	41.3

[1] Source: "Estimated Number of Survivors in Hiroshima City in 1950," Preliminary Report, Death Certificate Survey.

[2] Source: Listing of Leukemia Cases in Hiroshima and Nagasaki, September 1955. Cases are restricted to those in persons resident in Hiroshima at the time of diagnosis, and described in the listing under the heading, Diagnosis Acceptable.

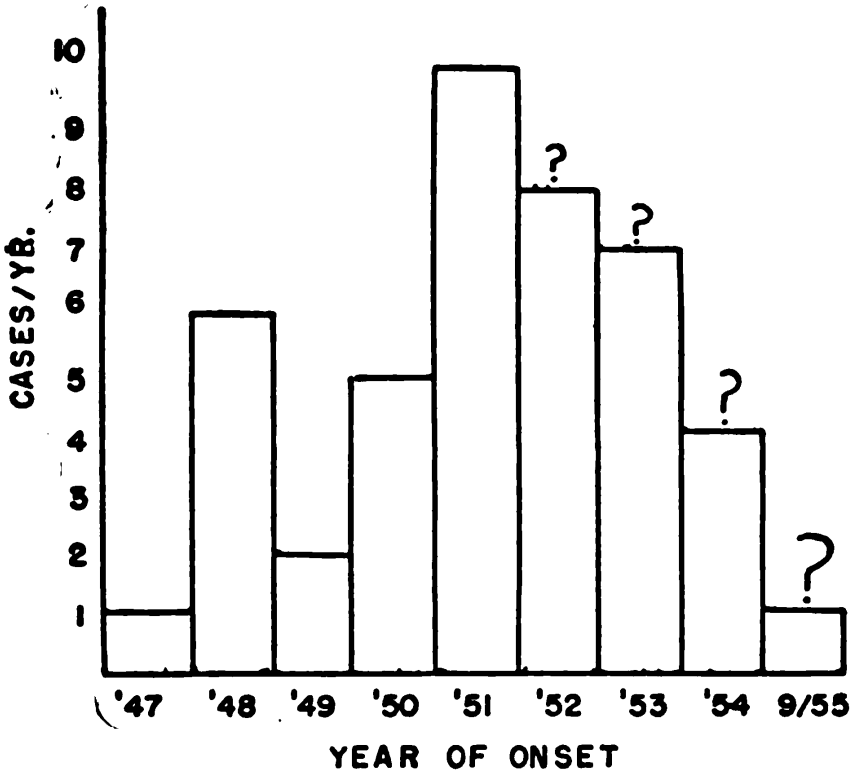
Leukemia rates in USA as listed on the 1951 record of Vital Statistics

	Male	Female	Overall rate
White	7.6	5.3	6.1
Nonwhite	4.0	2.7	----------

LEUKEMIA CASES FROM LISTING OF SEPTEMBER 1955

(Diagnosis acceptable - patients resident in Hiroshima at time of diagnosis)

ONSET OF LEUKEMIA



Note: Patients' histories not equally reliable. New data more likely to change totals for more recent years.

DEATH FROM LEUKEMIA

Note: 10 patients still alive. Their deaths may form another peak in next year or two.

U. S. Coast Guard. The AEC has worked closely with these agencies and is presently engaged in studies designed to minimize hazards incidental to future transportation requirements of the nuclear energy industry.

The relationship of nuclear facilities to basic public health, social and economic situations is a broad question upon which many studies are being brought to bear; for example, the Joint Committee on Atomic Energy established in 1955 a panel of prominent persons from many professions to study the impact of the peaceful uses of atomic energy. The report of this panel was published in two volumes in January of 1956. The Joint Committee on Atomic Energy and other committees of Congress such as the Committee on Interstate and Foreign Commerce, House of Representatives; the Committee on Government Operations, House of Representatives; and the Senate Committee on Foreign Relations, to mention but a few, haxe from time to time held hearings on various aspects of this subject.

This subject has also been of interest to many other groups including the Atomic Energy Commission, the U. S. Public Health Service, the National Academy of Sciences, the World Health Organization, the United Nations, the International Labor Organization, the Rockefeller Foundation, state health organizations and many other groups. These interests are developing our knowledge of the relationship of nuclear facilities to public health more rapidly than any other aspect of public health. Social and economic impacts arise more spontaneously from economic interests.

In reference to the last paragraph of Mr. Lorentz' letter, we believe that he is in error in assuming that Congress approves individual research projects rather than approving budgets for research programs.

We trust the information supplied above will be of assistance to you in replying to Mr. Lorentz' letter. Mr. Lorentz' letter is being returned to you.

Sincerely yours,

R. W. COOK, *Deputy General Manager.*

Enclosure: Letter dated April 1, 1957.

UNITED STATES ATOMIC ENERGY COMMISSION,
Washington 25, D. C., July 9, 1957.

Hon. CARL T. DURHAM,
Chairman, Joint Committee on Atomic Energy,
Congress of the United States.

DEAR MR. DURHAM: The Atomic Energy Commission approved for unclassified publication the rewritten handbook entitled "The Effects of Nuclear Weapons," copies of which are attached.

New information has been developed since "The Effects of Atomic Weapons" was last published. At the request and with the assistance of the AEC, the Armed Forces Special Weapons Project of the Department of Defense prepared the new edition. The services of Dr. Samuel Glasstone were secured as editor and the material was reviewed by the Federal Civil Defense Administration, by cognizant contractors of the AEC, and by the Department of Defense.

"The Effects of Nuclear Weapons" updates information appearing in the 1950 handbook and the February 15, 1955 release on Effects of High-Yield Weapons. It includes an expanded coverage on fallout referred to in our letter to Senator Anderson dated November 16, 1956. It bears a foreword signed by the Secretary of Defense, Charles E. Wilson, for the Department of Defense, Administrator Val Peterson for the FCDA, and the undersigned for the AEC. The handbook has been printed by the Government Printing Office which also prepared the advanced page-proof copies of those chapters dealing with fallout which were furnished to Representative Holifield of your Committee for the recent hearings on fallout.

Approval of the text by the Commission was unanimous except with respect to a number of points on which Commissioner Murray desired wording or action differing from that believed most appropriate or factual by the other members of the Commission. Commissioner Murray asked that you be informed of the points concerned; these are listed in the second attachment and comment thereon will be provided if requested.

These copies of the Effects of Nuclear Weapons handbook are being sent in advance of the release for the book which is set for publication in afternoon newspapers of Friday, July 12, 1957. When these copies were received it was

noted that the printer had omitted six lines from paragraph 10.24 on page 454. A correction sheet for that page has been included in each book.

Sincerely yours,

LEWIS L. STRAUSS, *Chairman.*

Attachments: As stated above.

Listed below are seven suggestions regarding "The Effects of Nuclear Weapons" handbook, which were made by Commissioner Thomas E. Murray but were not accepted by the Commission.

(1) That Chapters 9, 10, and 11 be submitted to the Advisory Committee on Biology & Medicine for review prior to approval for publication.

(2) That there be inserted in the Handbook a detailed Appendix of latest statistics and opinions relative to world-wide fallout.

(3) That in the last sentence of para. 9.49 which reads: "In fact the external radiation produced by the fallout from a weapon with a fission yield in the megaton range would be extremely small in comparison with the natural background radiation," the word "extremely" be deleted and, following the word "small," (of the order of —— percent)" be added.

(4) That in para. 9.94 of the Handbook, the words "can under some conditions" be replaced with the words "could be expected to" in the sentence which reads: "One is that the residual nuclear radiation can under some conditions represent a serious hazard at great distances from a nuclear explosion, well beyond the range of blast, shock, thermal radiation and the initial nuclear radiation."

(5) That in para. 10.1, the word "will" be used instead of "may" in the sentence which reads: "During the first few days or weeks after the detonation, the radiation levels may be high enough to represent a danger to exposed persons."

(6) That Chapter 10 entitled "World-Wide Fallout and Residual Radiation" give added coverage to the long-term strontium-90 hazard from local fallout.

(7) That in para. 11.122 the word "may" be deleted and the word "find" be changed to "finds" in the second sentence which reads: "The strontium may then find its way, mainly through milk products, into the human body."

APPENDIX 7

1. INFORMATION FURNISHED TO THE JOINT COMMITTEE BY STANLEY H. CLARK, BALTIMORE, MD.

2. INFORMATION FURNISHED TO THE JOINT COMMITTEE BY ALDEN A. POTTER, BETHESDA, MD.

8420 LOOKOUT MOUNTAIN AVENUE,
LOS ANGELES 46, CALIF.,
February 22, 1957.

Mr. GRAHAM DUSHANE,
Editor, Science,
1515 Massachusetts Avenue NW.,
Washington 5, D. C.

DEAR SIR: The article on Sr-90 content in human beings by J. L. Kulp, et al. (*Science*, February 8, 1957) may possibly be interpreted by some readers as indicating that there is little danger at this time from Sr-90. Actually that is very far from the truth.

First, consider the International Commission of Radiological Protection's recent (November 1956) reduction of the maximum permissible dose. It is now considered to be one-third (⅓) of the former value or five roentgens per year (this is occupational). Next we must always consider the occupational maximum permissible concentration separately from the maximum permissible concentration for the entire populace. The accepted reduction factor is ten—that is, the populace maximum permissible concentration should not exceed 0.5 roentgens per year (5×0.1). Applying to the Sr-90 maximum permissible concentration, we see that the populace m. p. c. is 33.3 micromicrocuries gram of calcium rather than the 1,000 micromicrocuries/gram of calcium as stated in the above-mentioned article. In the light of this value let us look at the actual Sr-90 content in human beings from different locations on the earth.

We see that the *average value* which is present in human beings today is only 1/330 of this m. p. c. and will become 1/33 (to 1/16) of the populace m. p. c. by

1970 even if no further nuclear devices are exploded! Now let us look more carefully at the concentration of Sr-90 in some of the 600 specimens analyzed. We find that 13 out of these 600 specimens actually exceed 1 micromicrocurie per gram of calcium *now* (through 1955). Or that they will have 10 to 20 micromicrocuries per gram of calcium by 1970 even though no further tests are conducted! These 13 specimens represent about 2 percent of the specimens which, if applied to the world population, is about 50,000,000 people! It will be noted from Fig. 2 of the *Science* article that these are mostly young people, 20 years or younger.

Of course, if tests are conducted at the same rate as in the past ten years, these 50,000,000 people on the surface of the earth would almost certainly be carrying an amount of Sr-90 equal to or even greater than the populace maximum permissible concentration of 33.3 micromicrocuries/gram of calcium!

Finally, we should note on page 68 of the British Research Council's Report "The Hazards To Man Of Nuclear And Allied Radiations" the following paragraph "In the light of knowledge at present available, we should feel that immediate considerations were required if the concentration (of Sr-90) in human bones showed signs of rising greatly beyond *one-hundredth* of that corresponding to the maximum permissible occupational level."

I believe that this information merits the attention of scientists and public alike.

Sincerely,

STANLEY H. CLARK,
Medical Physicist, Cedars of Lebanon Hospital, Los Angeles, Calif.
NUCLEAR DIVISION, GLENN L. MARTIN CO., BALTIMORE 3, MD., *March 15, 1957.*

10 EDGEVIEW RD., BALTIMORE 4, MARYLAND,
June 10, 1957.

Re hearings on the problem of radioactive fallout from nuclear weapons explosions.

Representative CHET HOLIFIELD,
Chairman, Subcommittee of the Joint Committee on Atomic Energy,
Room F-88, Capital Building, Washington, D. C.

DEAR MR. HOLIFIELD: I would like to submit for inclusion in the Congressional Record my feeling, as a scientist-citizen, on the subject of fallout and radiation injury. I would also like to make a suggestion for resolving the current conflict regarding testing of nuclear devices.

* * * * * * *

Although the medical genetic radiation exposures do exceed the fallout genetic radiation exposures in the United States, these medical exposures are knowingly received under medical supervision. This is not the case with world populace when we consider exposure from radioactive fallout. This, I feel, is the main point—we cannot expect all people to accept biological damage no matter how small or well justified we feel as a nation in exposing ourselves. *If there was general agreement* in the scientific community that there was negligible damage then such exposure would probably be acceptable.

Since the government has clearly stated that the testing of nuclear devices must be continued if our national security is not to be jeopardized, then we should look for solution to this problem which will not appreciably hinder future testing programs. The currently considered solution, that of reducing world nuclear testing to a few megatons per year, obviously does not fulfill the national security need. We must provide a means for testing nuclear devices by other nations as well as ours since world wide development of nuclear devices seems inevitable.

Probably the only solution to this problem is to make tests outside of the earth's atmosphere, i. e., in space. Today, 1957, this is technically feasible. We are now in a position to launch such nuclear devices on missiles that go well above the atmospheric envelope. I will not belittle the test difficulties; the difficulties in obtaining detailed test information; however, I feel certain the problems in this area can be resolved. Certainly the magnitude of an explosion and most of its physical characteristics can be ascertained during detonation in space.

The point I would like to stress technically is that it is completely safe from the radiological hazard standpoint to test weapons in space. (Probably the most serious hazard would be the great light intensity produced during such

tests.) What happens to the radioactive fission products when a nuclear device is exploded, for example, at 100 kilometers? Particles of 2 microns diameter (1 micron equal 1/10,000 centimeter) take a little over three years to fall to the earth's surface from this altitude. Particles of greater diameter take much less time to come to earth (at 8 microns diameter a particle takes about 130 days to reach the earth's surface). However, when the particle diameter approaches 0.2 micron or less, a new phenomena begins to occur. The light from the sun actually will push the particles out of the earth's gravitational field and, in fact, out of our solar system. Thus, for nuclear explosions in space with all particle sizes probably occurring of less than 1 micron, we have a means of cleaning up the fission products; in fact, sweeping them out into space. Surely this is the ultimate in a radiologically safe testing program. It is my hope that this means of resolving the nuclear testing debate will be investigated. If greater costs are necessary for such tests I feel certain that they will be jusified in the light of human well being.

STANLEY H. CLARK.

MEDICAL X-RAY EXPOSURES—NATIONAL VARIATIONS, INTEGRAL DOSES, ETC.

Stanley H. Clark,[1] National Biophysics Conference, Columbus, Ohio, March 4–6, 1957

INTRODUCTION

The development of the uses of atomic energy has focused greater and greater attention on the biological effects, particularly, the effects on man, of all ionizing radiation. The relative furor over the biological effects on man of radioactive fall-out has led to the refocusing of attention on existing and accepted uses of ionizing radiation. Thus, the pressures exerted due to new problems associated with uses of nuclear materials has caused a re-evaluation by a U. N. Committee, by Government bodies, scientific organizations, and individuals, of man's total radiation environment. Most, or all of these studies have indicated that of all the "men created" radiation exposures, that the uses in medicine are currently the most significant. Thus, we find that in the United States National Academy of Science Report, "The Biological Effects of Atomic Radiation" and in the British Medical Research Council Report "The Hazards to Man of Nuclear and Allied Radiations," as well as the U. N. Report on the biological effects of radiation that the medical uses of radiation have been subject to rather critical analysis.

Here is a comparison of the genetic exposure values in the medical uses of radiation as stated in the various national reports.

Values are for the first 30 years of life, and assume exposure at the same rate as at present and in the immediate past.

1. The United States as given in the National Academy of Sciences Report—3 roentgen.
2. The British, as given in the Medical Research Council Report—0.6 roentgen.
3. Sweden—Sivert's Study—0.78 roentgen.
4. Australia—Martin's Study—0.304 roentgen.
5. The U. N. Radiation Studies Committee Report—not yet completed wide range of values for different countries.

Let us now see what factors could account for these differences. (There is no particular order in this listing of factors.)

(*a*) Differences in actual number of diagnostic exposures to the populations of the various nations is one of the most important factors. Since each report sums medical exposures and then divides this lumped sum (in roentgens) by the numbers of people it makes considerable difference how many people in a populace have how many exposures. Related to the number of exposures per populace is no doubt standard of living of that particular nation.

(*b*) We might also ask, what medical uses are included in each nation's report. This calls attention to the fact that only the United States value includes therapeutic uses of X-rays (for non-malignant conditions), and uses of radioisotopes in medicine. These uses account for about 15% of the 3 roentgen value in the National Academy figure.

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[1] Cedars of Lebanon Hospital, L. A., Consultant, Medical Division, Oak Ridge Institute of Nuclear Studies, Physicist in Radiology, Medical School, University of Southern California, now with the Glenn L. Martin Company, Nuclear Division, Baltimore 3, Maryland.

(*c*) Difference in physical factors and techniques represents a significant variable. There is considerable more control of physical factors in England and in Sweden due to a combination of factors—socialized medicine; physicists have a closer relationship with radiologists; and early recognition of radiologic physics as a profession. In the United States one finds the private physician performing fluroscopy as well as other medical specialists, dentists, chiropractors, general practitioners, etc., using X-rays with few exceptions without any consultation with a radiologic physicist.

(*d*) Another factor that should be remembered is that of the origin of the various data which were used for averaging.

1. The National Academy averaged from a few United States hospitals and medical groups, however, correlating this data with nationwide uses of X-ray film, etc.
2. The British used hospitals in England and Wales which have hospital physics, groups and extrapolated for the rest of England. "Gonadal doses are based primarily on 1500 patients at one large hospital, (where incidentally particular care is taken to reduce the gonad dose to the minimum)."
3. In Sweden, most of the medical uses of radiation were considered in obtaining the average genetic exposure, no doubt the study was strongly influenced by the Institute of Radiopsysique in Stockholm where procedures are under direct supervision of a physics group.
4. In Martin's report on gonadal exposure due to diagnostic uses of X-rays he uses almost all available literature and the ratios of various diagnostic procedures in one hospital are the basis for his dosage estimates.

(*e*) Additional explanatory notes on factors which contribute to the range of exposure values.

1. The British Medical Research Council figure is really not comparable with the United States value since—to quote from that report—"The value of 22% (0.6 roentgen) should be regarded as a probable lower limit rather than as an estimate. A realistic estimate of the radiation contribution from diagnostic radiology might be considerably greater than this figure". They also discuss the possible factors of 2, 3, and even 10 times this lower limit.
2. In the Australian exposure analysis, J. H. Martin states "that the turn over of patients in the X-ray diagnostic department had already doubled" at the time of his presentation (November 1954), thus his values when brought up to date would be similar to, or greater than, the values for Great Britain and Sweden.
3. S. B. Osborn, whose work forms part of the basis for the British Medical Research Council's estimate discusses in his analysis in Lancet that examinations of the hip and lumbar spine, pyelograms and pelvimetry, although they constitute only 7% of the total number of examinations, none-the-less contribute 75% of the total genetic dosage. Obviously, even minor variations in these procedures from one hospital to another, would cause significant differences in the genetic exposure values. It is interesting to note that Osborne finds that 26.3% of the total populace genetic exposure takes place during radiographic exposure of the fetus in pregnant women.

What incident values mean in terms of whole body effect—the induction of cancer and the shortening of life span.

(*a*) There is need for the integral dose concept when evaluating the medical uses of X-rays. Discussion of radiation exposure of the gonads in man and the ovaries in women require measurements in the vicinity of the reproductive organs and some correction in women for the depth of the ovaries, however in this case we are concerned with the dose at essentially a point and need not be concerned with the energy absorbed in the entire body. However when considering the hazard from diagnostic uses of X-rays which may produce damage in terms of more general tissue damage we need a different concept. The concept most appropriate for evaluating such general body injury is that of the integral dose, or absorbed dose. Specifically, the two important effects related to integral dose are increased incidence of leukemia and shortening of life span. Both are known to be associated with exposure to ionizing radiation.

It is generally agreed that shortening of life span is strongly correlated with the amount of radiation energy abosrbed in the body. This effect then can be most appropriately discussed in terms of the integral dose—a concept originating in England, I believe—it is simply the product of the number of grams or cubic centimeters irradiated, and the number of roentgens to each of the cubic centimeter volumes. Such values are calculated from either X-ray distribution patterns known as isodose patterns, or from an equation developed by Johns. (6)

In general, the first technique is the more accurate. However, for the sake of our calculations, the John's equation has been used to obtain integral doses. When isodose curves were used the area between each isodose line was measured, using a planimeter. This area was then multiplied by the field height and the average roentgen dose to that volume to give the integral dose for that segment. These segment values were then summed to give the total integral dose.

Before comparing particular values of diagnostic integral dose, let us consider how the integral dose changes with change in the energy of the incident X-ray. Assuming that field size and exit dose-rate is kept constant how does the integral dose vary?

The integral dose decreases rapidly with increased X-ray kilovoltage assuming a constant exit dose.

Now turning to the diagnostic usages, we note that the patient exit dose must be essentially constant, regardless of KV, in order to produce useful film darkening or fluoscopic light intensity. This fact when considered in conjunction with the area under the depth dose curve (which is proportional to the integral dose) shows very clearly that in the range from 40 KV to 1 mev that the integral dose decreases substantially with increase KV—thus, we might ask why the diagnostic radiologist does not continue to raise the X-ray KV and thereby reduce the integral dose. There are at least two physical reasons why this is not wholly feasible:

1. The absorption coefficients for different elements in the body, i. e. bone and tissue become so similar at higher kilovoltage that very little contrast is obtained at energies above a few hundred kilovolts.

2. The response of film (and fluorescent screens) decreases with increase KV for a given incident intensity. It becomes evident that there are optional KV values for various parts of the body, depending strongly on what information one is most interested in and the thickness of the particular body cross section. It should be remarked that if too much filtration is used at high energies there will be no reduction of integral dose. There will be a decreased incident dose, but the exit dose must be increased because of the decreased film response at higher KV.

(*b*) Since the increased incidence of cancer, primarily leukemia, is related to the actual bone dosage as well as to the integral dose, it is important to note the depth dose distribution as well in evaluating this particular hazard. It has been pointed out by Hardin Jones and others, that the dosage to long bones, i. e., the dosage to the rib cage, in chest X-rays would be particularly important insofar as the production of leukemia is concerned. It would be well to mention at this point the studies that indicate leukemia is produced by even small amounts of radiation. The best evidence is from the following studies:

1. Studies by Alice Stewart, J. Webb (7, 8), etal in England which indicate an increase in incidence of several malignant diseases including leukemia due to diagnostic X-ray exposures of the pregnant mother, particularly abdomenal exposures. Secondly studies of the survivors of the Hiroshima and Nagasaki nuclear explosives (9, 10), and thirdly exposure of radiologists (11) in the course of their professional activities.

2. Studies which indicate that somewhat larger radiation doses will produce leukemia include a series of infants treated with X-rays for thymus condition (12) and secondly X-ray therapy of anklosing spondylitis (13).

The statistical analysis of this information, as well as the linear relationship between incidence of leukemia and integrated dose is given strong support by Hardin Jones of University of California, in material which will be published in the near future. Shortening of life span as caused by radiation has been borne out by many animal experiments by the shortening of life span of radiologists, and by patients who have been treated with X-rays. Here again, the dose is linear with respect to shortening of life span. According to Jones about 10 days should be subtracted for each roentgen received of whole body radiation. The integral dose ranges from about 11,300 to 56,000 gram-roentgens for 1 roentgen incident dose. This assumes a range of X-ray energies corresponding to 2 mm of aluminum half value layer to 2 mm of copper half value layer.

In the course of using X-rays for treatment of malignant diseases, we encounter total integral dosages ranging from 1 million gram-roentgens to many million (30 or 40) gram-roentgens. This wide range is due primarily to the variation of field sizes that are used for lesions of different dimensions, and to some extent of KV. These large integral doses are actually admin-

istered in daily doses—as many as 30 or 40—in the course of a single treatment. To cite a specific example—using a 5 cm circular field at a focal skin distance of 80 cm with a half value layer of 4.00 mm of copper. The daily dose of 100 roentgen produces an integral dose of about 19,800 gram-roentgens, (remember that one roentgen incident dose produces about 11,300 gram-roentgens). Now let us compare this therapeutic value with some common diagnostic procedures. A film of the lumbar spine taken through the AP direction with the following physical factors—AP thickness 20 cm F. S. D.-71 cm, KV-70, MAS 160. Focal film distance 91.44 cm (36 in.) produces an incident dose per exposure of 4.3 roentgen. This produces an integral dose of 28,810 gram-roentgens. If we take a lateral film of the same person, we will obtain an integral dose of about 150,000 gram-roentgen or the equivalent of some 7½ times the daily integral dose cited for the above cancer therapy! Or to make another comparison, about 3 times the present maximum permissible exposure for one year period! This value would be greatly increased for a heavier person. The explanation for the larger integral dose as compared to the therapeutic dose is almost entirely due to the increase in field size. In the therapeutic example, the field size used was about 20 cm^2 in the diagnostic case it was 1487.5 cm^2 (a standard 14x17 inch film). Of course, the actual X-ray field size as determined by the cone used was circular and somewhat larger even than the area used. The integral dose in chest photofluoraphy would be about 10,000 gram-roentgens.

In fluoroscopy, the literature cites incident dosage rates from 5 to 20 roentgens per minute, depending undoubtedly on the amount of filteration and body thickness. This dosage rate would produce integral dose values from about 30,000 gram-roentgen per minute to about 120,000 gram-roentgen per minute. Frequently fluoroscopies last as much as 5 or 10 minutes which means the absorbed energy approaches that which is actually used for some small field cancer therapy. What does this mean in terms of shortening of life span and increased incidence of cancer? In the case of the shortening of life span we found that a 1 roentgen incident dose (which amounts to about 11,000 gram roentgen would decrease life span statistically by about 10 days. Thus, a single fluoroscopic examination would shorten the life span by as much as 500 days (at a 20 r per minute dosage rate). Or the lateral pelvic radiograph would shorten the average life span by about 140 days! But let us see how these figures compare with some other factors that are known to shorten life span (unpublished, Hardin Jones-University of California).

	Years
25 percent overweight group	3.6
Heart murmur	11
Rapid pulse	3.5
Varicose veins	0.2
Trace of albumin in urine	5.0
Epilepsy	20.0
Skull fracture	2.9

It should be noted that when applied to the total populace millions of man-years of life are lost due to medical radiation exposure (about 7,000,000 man-years each 30 years at the current exposure rate).

With respect to increasing the incidence of cancer particularly myeloid leukemia I would simply like to make a general statement regarding the increased incidence with respect to chest photofluography as a significant example. We find that 1r exposure per year gives a probability of 1 in 100,000 to 1: 1,000,000 of developing leukemia per average individual (7) (14) (15). This probability applied to the 15,000,000 people in the United States who have chest X-rays (in mass chest X-ray surveys) annually would amount to an increased number of cases of myelogenous leukemia; due to these chest X-ray exposures alone 15 to 150 cases per year and each year thereafter. There is in addition speculations that some individuals are genetically more asensitive to the induction of radiation leukemia. Thus a particular sub-population might have a considerably higher probability of the occurrence of radiation induced leukemia.

MISCELLANEOUS-CONCLUSION

Finally let us consider the exposure to X-ray technicians in the course of their training, there are some 40,000 in the United States. They divide up into pairs

and go through the entire radiographic series of exposures normally encountered in diagnostic X-ray work. The genetic exposure for 35 procedures amounts to about 5.6 roentgens for the male technicians and 6.3 roentgens for the female technicians. This exposure corresponds to perhaps 20 to 30 roentgens incident dose (not greater) or about 10 to 15 roentgens of whole body radiation (in terms of integral dose). Thus in this group of technicians due to their X-ray exposure during training we might expect (using a probability of 10^{-4} per roentgen per year) some four cases of radiation induced leukemia per year. The additional exposure in the course of their work is not considered. Even so, since X-ray technicians are generally young people (at the time of training) we would expect this increased incidence of radiation induced leukemia to amount to about 160 additional cases (normally one would expect about 240 cases of both lymphatic and myelogenous leukemia). Hardin Jones states that the leukemia doubling rate is about 30 roentgens full body exposure. Which would give a figure of about 120 cases of radiation induced leukemia. It is hoped that this estimate could be confirmed from actual death statistics of X-ray technicians.

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BETHESDA 14, MD., *July 5, 1957.*

Hon. W. STERLING COLE,
Joint Committee on Atomic Energy,
Washington, D. C.

MY DEAR CONGRESSMAN COLE: Through your kind services I beg to submit the enclosed "Critique of Scientific Knowledge" for entry, as the Committee may see fit, in the record of the hearings on The Nature of Radioactive Fallout and Its Effects on Man, held in May-June.

The Kremlin seems to have shifted its position, softened its opposition to "western" genetics which they have ardently rejected—until it came handy in their cold war in Japan against *our* bomb tests.

At the suggestion of the Committee Technical Advisor I am asking the following geneticists with whom I am acquainted to comment, to the committee on copies sent them.

Dr. James F. Crow, National Institute of Genetics, Mishima, Japan.
Dr. Carl C. Lindegren, Southern Illinois University, Carbondale, Ill.
Dr. Stanley H. Emerson, Atomic Energy Commission, Washington, D. C.

Very truly yours,

ALDEN POTTER.

A Critique of Scientific Knowledge

Keep that which is committed to thy trust, avoiding profane and vain babblings, and oppositions of science falsely so called (I Timothy 6.20).

The evidence adduced in these hearings on the effect of radioactive fallout on human and other life has shown that even a considerable enlargement of test explosions will not increase the incidence of radioactivity materially beyond what all forms of life have experienced from time immemorial.

A prime consideration is the growing belief that most of the radioactivity that is injurious can be eliminated from military weapons by tests now being planned and conducted, so that possible danger to future generations in case of war will be correspondingly reduced. The larger, fusion bombs are less radioactive in proportion to the success that may be attained in eliminating fission, as in "triggering" the explosion. This may explain the Kremlin's current readiness to suspend tests so they can catch up with our "classified" progress toward clean bombs, while shedding crocodile tears over the genetic horror which they have subtly helped "western" geneticists implant at the Japanese National Institute of Genetics at Mishima.

The attempt to make medical science "objective" has turned on statistical theory and practice which is leading to a great deal of confusion as to the applicability of degrees of probability thus determined. This source of misinformation is by no means confined to research in radioactive fallout as presented in these hearings, but extends over the whole field of science. It will therefore be necessary to attempt some clarification of these basic disputes as they affect the argument over whether observed effects may be linearly extrapolated (whether the extent of biological injury by radioactivity is directly proportional to the extent of such activity, however small), as against the existence of a "threshold" or range of radiation that is either beneficial or at worst not injurious to any life. This critique will contend that thresholds are the rule, not the exception, in nature. Moreover, insofar as a threshold is biologically determined, we shall argue that it cannot be a fixed factor in the survival of any species because of adaptive changes in the organisms involved.

Senator Anderson has cited such an adaptation in the case of the resistance of insects (flies) to DDT; and there are many other such cases in recent experience with insecticides. This factor of adaptive change has been so widespread in medical research that in 1954 the University of Pennsylvania's medical school cooperated with Naval Research in organizing a symposium on drug resistance at the Statler Hotel in Washington; and thereby hangs a tale. The honor guest and speaker at dinner was Dr. Cecil P. Martin of McGill University in Montreal, Canada. He was called upon to speak because of an essay, "A Non-Geneticist Looks at Evolution," published in the American Scientist shortly before, in which he assailed "Western" genetics on the ground that it has produced an untenable theory of evolution, that is, the mutation-selection theory.

This view has gathered some support even among geneticists; witness the recent note (Science, May 10, 1957) of Lindegren and Braun criticizing the views of Dr. George Beadle of the California Institute of Technology as given in his presidential address to the American Association for the Advancement of Science, for his too confident proposal to call nucleic acid a "living" molecule because it is a "carrier" of genes in heredity. The essence of this question lies in the way the species pattern is carried. Is it by a template—a minute replica as Dr. Glass contends—or is the system like our cultural information system, linguistic and therefore *meta*physically informational like an engineer's handbook?

It should be unnecessary to record the views of Dr. Martin here for they have been available in a treatise published last year by the eminent medical publishing house of C. C. Thomas of Springfield, Illinois, entitled "Psychology, Evolution, and Sex." But the evidence adduced in these hearings by the panel of geneticists from the National Academy of Science has studiously omitted any reference whatever to these conflicting opinions, quite as they have been omitted from any and all proceedings and reports of the Academy and, as to the Martin treatise, also from the pages of Science where the book has not been reviewed after almost a year since its publication.

It will be necessary, therefore, to try to clarify the relation between the Darwinian views of Dr. Martin and some few geneticists whom our Academy has deliberately ignored, and those set forth by the Soviet scientists at a recent conference in Japan on genetics where they spoke in the terms of Mendelian theory for the first time since the days of Trofim Lysenko. Is it indeed a mere coincidence that the mechanistic materialism of our Academy with its pretense of

"objectivity" now serves in furthering the curtailment of bomb testing just as the Russian cold war is so intensifying its peace offensive as to produce riots before the American embassy in Tokyo? Are we not ourselves so supersaturated with the prevalent anti-Darwinian philosophy that we refuse to examine such a scholarly treatise as that of Cecil Martin which counters the false alternatives of an alleged conflict between military and genetic security? Only the mutation-selection doctrine with its elmination of competitive stratagems in organic evolution can serve the communistic pacifism of Soviet propaganda; and it is with a sudden acceptance of this idea that they carry the war into Tokyo!

A clarification of these issues on an honestly open-minded, objective basis entails a thorough reconsideration of the basic tenets of *scientific empiricism* which define "the scientific method" ("Scientism" and "positivism" are other terms for this "unity of science" philosophy) as purely inductive. Such a reconsideration is supported by the rising tide of literature indicating the imminence of a conceptual revolution in science. This deductive revolt is renewing the conflict between science and the anthropocentric bigotry which gave rise to the Scopes trial in Tennessee instigated by William Jennings Bryan. This court case sought to ban the teaching of a theory of evolution that does not admit the uniqueness of man which is today propounded by the chemical theory of genetics and the origin of life because, very obviously, *human affairs are certainly not chemically explicable.* Man does not live by bread alone; *but neither does any other form of life.*

The bigotry of this anthropomorphic theology has unmistakably injected the pontifical edict that an "emergent" evolutionary theory may be anthropomorphic upward *to* divinity, but not upward *from* monkeys, in its "extrapolations." Our Fundamentalists thus create a self-idolatry—a God in the image of man; an authoritarian threshold that may not be crossed even by the authorities. This prideful conceit that man is peculiarly unique not only has no support in the Christian Gospels but it seems to beget a peculiar predilection for statistical extrapolation, while banning biological analogy, as scientific evidence.

Mice are not men; so science must perforce extrapolate averages (statistical data) as a "first approximation" or "educated guess," the while indulging in crash projects, not to test empiricism against an alternative postulate, but to "prove" that extrapolations (predictions) are correct and so justify, by pragmatic "proof", the public policy already adopted in "playing safe" with probabilities. This is called "verifying the hypothesis" with "observational evidence." Its successes are proclaimed from the housetops; its failures suppressed like a Nobel Prize gone sour.

In assailing this materialistic biology it would be well to cite further evidence, from outside these hearings, that it does exist and is, indeed, censoriously entrenched in scientific literature and training for "academic freedom" in research. The popularity among biological empirics of a small book written by Prof. Erwin Schrödinger, author of the principal equation of the so-called "quantum mechanics" of atomic physics, "What Is Life?", with its attempt to forecast a physical answer, is attested not only by numerous reprints (including a paperbound, popular edition) but also by the widespread opinion, urged most recently by Linus Pauling in a lecture at the National Institutes of Health in Bethesda, that the genesis and genetics of life can and ultimately must be defined in purely chemical terms. Any other outcome would, *ex cathedra,* be a betrayal of progress in "science."

Since these hearings were recessed early in June, an article has appeared in Science (June 7) based on another lecture at CIT (alphabetics for California Institute of Technology) by an Iowa biophysicist (Robert Sinsheimer) on "First Steps Toward a Genetic Chemistry." Here "steps" in progress *toward* the end *sought*—toward what "we *shall* discover" chemically—are recounted as if time were reversed by some extrasensory perception. The facts are "not a sufficient proof * * * but in a way it is *satisfactory* to believe that the physical basis of the gene—the factor that is passed on from cell generation to cell generation—is physically conserved * * *." (Just as it was satisfactory to believe that "parity" was "conserved" in quantum mechanics—until it was shown, not first by observation, but by straight thinking, that it was untenable however satisfying!)

To our biophysicist certain chemical correlations have provided "a *pleasing* confirmation of our hypothesis," even though inconsistent with other facts. To get rid of these embarrassing obstacles to *pleasure* "more refined techniques could prove to be of *great value*" in the "*hope* that the development of a genetic

chemistry will *help* * * *." Why, indeed, are "elegant tracer studies" credited with a "*pleasing* result"? Why do "we know *painfully* little"?

Obviously because "we" are extrapolating our pullets—counting them before they are hatched. Come what may, "we" are not going to hatch any cockerels as long as any refining of techniques (or elaboration of hypotheses) remains to renew the hope that springs eternal in the chemical breast that we shall soon know how to sex them, not by the Japanese manual methods after hatching, but chemically, before the eggs are laid. Some day there will be an RNA for sex, chemically the same for all life from virus to vireo to mastadon.

Here, indeed, is an endless frontier for scientists; a fiscal rathole down which appropriations and empirical data can be poured endlessely before reaching any conclusions; such as foreign-aid policy for Japan to provide a chemically immaculate conception to displace the recently legalized abortions that are keeping Japan (and China, too) from being any further "under-developed"; that is, overpopulated. Why, indeed, worry about poverty if all we need do in the matter of a trait called parental care is to take the long last step in genetic chemistry, to wit, find the proper, parental test-tube in the gene-bank kept under lead to exclude radioactive fallout? We are definitely assured that there *is* a chemistry of the gene (not yet correlated with DNA chemistry, but soon to be) which, *when* "we have become sufficiently advanced in our understanding" of the raveled sleave of mitosis, *will* enable us to "demonstrate the action of a gene *in vitro*"—so we can live *in vitro* for nine months and then have plenty of fresh milk.

To force a reconsideration of the empirical "unity of science" is more of a task than can be encompassed by this brief critique. Such a reconsideration might, however, be invited by a rather categorical review of the high spots in this concaptual revolution as dimly perceived despite the din of data in the process of erecting our scientific Tower of Babel. Conceivably we might thus stir the well-known, open-minded curiosity of scientists in their zealous search for ways to "ring out the old; ring in the new; ring out the false, ring in the true." For not one but many minds are needed in the process of dismissing all the facets of fallacy involved in a postivistic "idealism" which obscures reality by making it synonymous with actuality so that "objective" means a consensus of *subjective* judgments as "verified by experience," pragmatically.

Life on Earth, as a whole, has no opportunity for the unlimited expansion in which it seeks to indulge. For this reason what men, and all other organisms, have most to fear is not the physical environment and its accidents, but far more what other biological occupants ("fellow creatures") do to that environment in the struggle to survive. What any and all biological coalitions do is not purely physical or fortuitous. It is expediently adaptive in its strategic choices so that all free-will, animal life requires eternal vigilance to choose aright and avoid extinction at the hands of other predators who are also exploiting other organisms as "renewable resources," sometimes symbiotically.

Among men, in a civilized context, Disraeli put the case for vigilance against misinformation in a retort to "factual" argument by the Loyal Opposition in parliamentary debate: "There are three kinds of lies; white lies, damned lies, and statistics." Since then there have been frequent protests against empirical "objectivity" among "experts" who employ "factual" correlations to verify hypotheses. These have pursued the pattern, reflecting Christ's warnings against false prophets set forth by St. Paul to Timothy telling him to avoid "profane and vain babblings, and oppositions of science falsely so called."

There have been side-splitting satires, like Chick Sales' "The Specialist," and, less biting but more specific, Anthony Standen's "Science Is a Sacred Cow" featured by the author's review, with lampooning cartoons, in LIFE seven years ago. Lately a physical chemist turned social philosopher, Prof. Michael Polanyi of Manchester, England, has severely indicated *scientific empiricism* in America in a paper read before a symposium on "Fundamental Concepts and Units of Science," published in Science this past winter.

Most recent and satirical of all is a contribution from Cornell University by zoology professor LaMont Cole, "Biological Clock in the Unicorn" (Science, May 3, 1957). This paper so aptly caricatures statistical correlation in verifying an hypothesis that it is astonishing to find, following it by only two weeks in the pages of Science, the same "quantitative information about the effect of radiation on human health" as that presented before this committee by CIT's Prof. Edward Lewis on "Leukemia and Ionizing Radiation" which Dr. Shields Warren rejected as inadequate evidence of causation in leukemia, supported editorially as follows (DuShane, Science May 17, 1957, p. 963, "Loaded Dice"):

"E. B. Lewis shows that there is a direct linear relation between the dose of radiation and the occurrence of leukemia * * * The meaning of such findings is that any amount of radiation takes its toll of the population and any increase takes a greater toll * * * We are approaching the point at which it will be possible to make the phrase 'calculated risk' for radiation mean something a good deal more precise than the 'best guess.' It is apparent that the atomic dice are loaded. The percentages are against us and we ought not play (with bomb tests) unless we must to assure other victories."

The movement toward clean bombs for the West which was revealed in these hearings and later publicized by the President (and caustically condemned by the Kremlin!) has taken the wind out of the Schweitzer sails (and the Pauling Petition) as related to leukemia and strontium 90 so that what remains of the loading of atomic dice by statistical correlation is now confined to genetic effects as alleged by the panel from the National Academy of Science before the Committee on June 4.

If it is not true that (DuShane) "much can be learned about biological reactions (in man) by appropriate statistical and epidemiological studies," such as that of leukemia conducted by a geneticist and statistician (Lewis) who has the "quantitative information" but not the facts about the disease (the recent rise in the statistical incidence of leukemia has been due, not to improved methods of diagnosis, much less to any increase in casual factors, but to the use of penicillin and other "wonder drugs" to save the patients from infections to which bad blood makes them very susceptible), it is equally fallacious to conclude that a correlation between chromosomes and the statistical results of sexual crossing reveals the true character of survival values in organic evolution.

By the same token it is not competent to conclude from a "linear relation," as did Professor Crow before the Committee, that "death, disease, and misery" produce "natural selection" without considering the occasion for such phenomena; for example, war, famine, and pestilence among men, which would be extremely difficult to correlate with any genes without even greater stretching of hypotheses than has as yet been employed in genetics. ("To multiply auxiliary hypotheses is to goropise." See "The Principle of Simplicity" by Lewis Feuer, Philosophy of Science, April, 1957.)

It is not because fruit flies are not men that the monstrous effects of laboratory irradiation do not prove the thesis that the human race will suffer from bomb tests for centuries to come. It is because mutations are not adaptations that this conclusion is unwarranted, either for men or for fruit flies. Gene injuries are as possible as any other anatomical damage from irradiation. But that they persist as recessives throughout a population (without benefit of the dysgenic effects of medical science among men), is not proved by selected statistical averages that destroy the facts.

Mutations, when they do occur, are baneful, indeed; for they destroy the hereditary homeostasis of the species in much the same sense that a burning of books can destroy a cultural heritage. They are *devil*-utionary, not evolutionary. Unlike adaptations to meet competitive pressures they do not remodel the species for survival and become, as Martin contends, "lingering modifications" which do evolve new genes in the course of unobservable time—time which is measured, not in terms of sidereal or sexual events (years or generations) but in terms of marginal elements in interacting stratagems that cannot possibly be reduced to "quantitative information."

Adaptive changes do not occur in detectable form for easy correlation; but they are not therefore mere accidentally adapted mutations. They are teleological expedients, intentionally produced, in the unobservable realm of the microcosm. This is, indeed, an unverifiable hypothesis. But it is not a "goropised" set of hypotheses devised to verify each other in an endless tautology; nor is it untrue because it is unamenable to empirical confirmation by "the scientific method," as was the case with a similar hypothesis in quantum mechanics, to wit, the conservation of parity, which was found to be *predictably* and *testably* false because it pertained to purely physical, inorganic structure. Not observation, but theory, made this discovery!

In biology theory must perforce pursue analogy—the judging of all living processes by our own, in aspects which every man-on-the-street knows and which require no expertise explanations; and these processes are not predictable in their course. They are expedient; just for today. While they cannot violate the natural laws of the physical order, they are not governed by them. They

are "cybernetically" governed, by negative feedbacks that involve a correction in the current theory of information—a corrective which postulates the existence of a metaphysical factor, that is, a dialectical system of commication analogous to human communication in everyday affairs wherein *conditioned* reflexes are a matter of the most familiar processes of modern education. As Whitehead has pointed out, the more civilized we are, the more automatized is our behavior. (Cf. Hayek, "The Use of Knowledge in Society," Am. Econ. Review, September, 1945.)

Concurring with the Martin thesis, Lindegren and Braun (Science, May 10, 1957) cite literature which holds that gene mutation *per se* does not afford a satisfactory explanation of evolution; also that the gene, like sex, is not primitive; nor is its alleged stability a fact.

Pertinent to the fallacy of genetics as a physical science is my own senior thesis dated 1909 (Minnesota) on "The Cytology of Weismannism." This paper not only rejected the mutation-selection theory in almost the same terms as those of Dr. Martin's treatise of 1956, but in a full review of the evidence in the literature of genetic cytology, nucleic acid was twice mentioned as playing some part in the processes of heredity. Though this theme of the early twentieth century is "dated" in some particulars, there is no more reason now than there was then to believe, with Sinsheimer, that "this hard-won recognition of the role of DNA (nucleic acid) has brought us into a new era in genetics and biochemistry. The gene, once a formal abstraction," he concludes, "has begun to condense, to assume form and structure and defined reactivity."

That this is wishful thinking; that there is nothing new in the evidence at hand today which can change the conviction that a chemical explanation of life can never be adduced, can be seen by comparing the two papers. So that interested readers may do so, "The Cytology of Weismannism" is here submitted for its first printing.

To predict by extrapolation from statistical correlations is the unforgivable sin of biological empiricism. There are some contexts in medical practice having legalistic implications, where statistically "educated guesses" as to "calculated risks" form the basis of "expert" opinion used, for example, in judging degrees of disability and causes thereof, as in insurance payments, suits for damages, social "security", veterans compensation, and the like. But in a social context where the results of taking chances with probability are compounded by a multiplier or even exponentially, as in public health policies or in the anarchy of foreign relations where victory or defeat in global warfare is the risk involved, its success, if any, is at best illusory and its failure catastrophic. Biologicaly and culturally Operations Research is worse than futile; it is fraudulent. In medical practice or research epidemiological figures can at best furnish only clues, never evidence that confirms any hypothesis. If the patient is going to die anyway—a fix in which everyone finds himself sooner or later—an experiment may be in order. But when the fate of a people is in the balance and survival is at stake, in disarmament schemes for instance, or when the health of millions of children is concerned with the standardizing of a vaccine, then a trust in statistical probability as against a laissez faire policy, can be disastrous.

Such an error has just been courageously exposed, even in Science (May 31, 1957), namely, the fiction of the safety of the Salk polio vaccine; nor is this the first Nobel Prize in medicine that has not been rescinded in the light of the pitiless truth as to its falsity. In this exposure of the untruths broadcast by the National Foundation for Infantile Paralysis, falsehoods which are plainly proclaimed, there is a serious omission in failing to state the underlying occasion for them, to wit, the idea that the degree of probability can be scientifically measured in biology as in physics and make (to quote the DuShane editorial on dice again) "the phrase 'calculated risk' * * * mean something a good deal more precise than the 'best guess'," and afford a reliable basis for a public policy based on "appropriate statistical and epidemiological studies."

That hundreds of children were not paralyzed and crippled, or even killed, was the work, not of medical science, but of nature; for had the same children been vaccinated with unattenuated virus very few clinical cases would have developed. The probability that infection will cause the disease is so low that its incidence cannot be taken as a measure of the spread of the virus. Poliomyelitis is a relatively rare disease; so also is leukemia. Vaccines for all such afflictions—and they are multifarious and but dimly classified—would burden the community beyond endurance if promoted as a public charity. The treatment of hog chlorea by a similarly attenuated, living vaccine has admittedly kept the disease active instead of exterminating it; so at long last the live

vaccine is being eliminated by law. It has been unjustified also in poliomyelitis, despite the full responsibility of the National Institutes of Health for promoting its use.

The logical fallacy of statistical biology with its reliance on correlations can be shown by an "anthropomorphic" analogy such as was banned from Tennessee public schools by law and, right now, is "scientifically" tabooed by the "positivistic" ban on "anthropomorphic extrapolations."

The cultural DNA of modern civilzation is plainly *money*. That it is coined from gold and other "precious" metals is as irrelevant to its value as an informational guide for human exchanges and consequent behavior, as is the forming of genes (genetic prices) from nucleotides. It is quite as absurd to believe that the substance of DNA must differ in its physical structure in order to create a man instead of a monkey, as it ever was to believe that an ovum is simply a small edition of an embryo and has only to grow to produce an adult man or monkey.

The assertion of Dr. Glass before the Committee that something like a template (a "mould" or "replica") is involved in the embryonic developmental process, is just this sort of untenable belief for which there is no evidence at all. At fault is, basically, the treatment of "information" as a matter of signals as distinguished from signs or symbols with a metaphysically conditioned system of meaning. It is not at all necessary for the same word (or figure) to be used to convey the same meaning (or value); nor different words to appear when different meanings are conveyed. Ambiguity is rampant in language—as are paradoxes in mathematics. An assumption that there is a similar dialectic in genetics constitutes the only possible alternative for the empirical ambiguities which must be eliminated from science by sound reasoning from realistic principles.

The composition of chromosones, genes, alleles, genomes, nucleotides—or what-have-you in genetic equipment—can never determine what they signify in the organism's behavior and development, just as monetary units of various denominations cannot determine the real values underlying the judgments and consequent behavior of a business community as guided by intelligence (price relations); the values are the same regardless of the prices which the number of dollars or pounds determines. *Changing* prices do distort the normal course of judgment and action just as ambiguity distorts the influence of speech or literature in behavior. The fact that it is an artifice, not a reality, that creates the uncertainty of meaning and of biological behavior in general through *conditioned* reflexes (learned symbols) is the essence of the problem of living together by communicated information. There are no puns or paradoxes in nature's realities.

If a Man from Mars were to observe the number of pounds in a British pocket, and the number of dollars in a Canadian pocket, he would have no way of knowing that they differ in their value even though they look alike. So if he were to use Cartesian coordinates in two dimensions to correlate these numbers with the energy level of observed events (behavior) related to those numbers, would he be warranted in "discovering" that this graphic relation is a natural constant? What should he conclude if the British set up another Newtonian Recoinage Commission, or if an International Monetary Fund changed its "mind" on the proper exchange rate, so that the energy level of events departed from the previously observed, linear correlation? Should he call it a "spontaneous" mutation? Would not some Martian jester turn up to publish a report in a Saturnian Science on the rhythmic clock of *Unicornus martius*?

Of Dr. Bentley Glass discovers (as he has) that the correlation between the traits of *Homo insipiens* and the number of chromosomes is not 48 after all, shall he conclude that all the observers who counted 48 were cross-eyed, or that their staining techniques were defective? Or should he conclude, with an ancient inscription in the Libyan Desert, that "Life is change; to cease to change is to cease to live"? If the nucleotides-to-be-counted with electronic relays when genetic chemistry gets a big enough appropriation to buy a big enough computer from IBM, turn out, as all such research to date has done, only some more negative results in the attempt to correlate their chemistry with their behavior in heredity, will it at long last become reasonable to agree with Dr. Lindegren that genes are not stable after all? Will it finally be admitted that his charge that the evidence of such stability has been a matter of choosing only data that support such an assumption, is warranted?

The loophole in the "laws" of Mendelian inheritance as presented by the geneticists before this Committee, lies in their manipulation of "spontaneous"

cases of genetic change to avoid admitting that any of them are adaptive. Adaptation has been ruled out of the vocabulary of organic evolution so that life is today supposed to be one grand symbiotic brotherhood seeking a communistic goal in selfless abandon. Perish the thought of conflict of purposes acting incompatibly in a tooth-and-claw struggle, or of any innovation in survival that has been other than the result of rare accidents in throwing genetic dice that are *not* loaded! Those big claws on Alaskan king crabs turned pink by our loving kindness, are purely ornamental, love-patting appendages, swords beaten into plow shares like atoms-for-peace in Utopia.

The dispute in these hearings over the interpretation of data has hinged on the idea of a threshold as opposed to linear extrapolation, rather than on the evidence that has here been adduced as to the absence of biological constancy where genetic science has postulated it. What remains to be shown, therefore, is that nature consists of thresholds, not of linear correlations, and that research consists of discovering the status of thresholds, never in tracing imaginary linear relations. This would be obvious but for the linear predilections of the Euclidean influence in science for even in the inorganic realm of physics the field of macrocosmic relations is not universal; witness phase transformations such as melting points and boiling points and their energy relations. A jet plane passes from one range of speed into another with an explosive sound as it crosses the "barrier" into a very different set of relations. Physical constants established by instrumental measurements (as by the National Bureau of Standards), such as those of Hooke's Law on the strength of materials, which are inapplicable to very small sizes such as exist in one of the most recent developments in solid state physics (metallic "whiskers"), are another example.

In biology the operation of sensory perception, from which all knowledge proceeds, is replete with thresholds. The *source* of knowledge is the same for any and all of the creatures that inhabit the earth; it lies in a myriad of wave forms, or bands, filling all possible environments throughout the universe. But there are great areas of this spectral information that are extrasensory for any mammal. Some bands are accessible to insects but not to vertibrates; and some mammals such as rats, bats, cats, dogs, can sense ranges greater or less than other species, man included. Some men can sense a range wider than others; but none can be devoid of all sensation and still live.

Other sensations can be translated into tactual perception, as by Braille, to make individuals "literate", that is, to train their reflexes for communication purposes in guiding behavior. In this aspect of a cultural heritage as it operates genetically—and genocidally—in the human struggle to survive, telecommunication preforms transformations in and out of sensory ranges, with continual improvements for technically conditioned people.

Thus science as we know it is continually extending the range of sensation and communication to new bands and ranges; but these new, extrasensory signals have to be modulated into the normal sensory ranges or thresholds before they can affect behavior. No possible information can be had without reference to these physical wave bands as they have been affected by discontinuities (things) in the pyhsical universe. It is because the laws governing these spectral conditions never change anywhere in eternity, so that the sequence of events they reveal is absolute, that time cannot be reversed or events known that have not yet occurred. (Cf. Anthony Standen on "Causes and Effects" in Science, May 3, 1957, p. 900.) Thus no possible living creature can have any information available for conditioning its reflexes (guiding its behavior) that does not rest on these ordered discontinuities (waves and particles), in the physical environment, that are the source of all certainty in sensory experience.

But if sensory equipment has its thresholds, living conditions are also narrowly restricted to those prevailing in the so-called biosphere. Some heat is essential; much more or less, is lethal. It's a case of not crossing a *threshold* and getting *too much* of the good things of life; including life itself. To avoid too much or too little, animals have evolved sensory equipment which men are still evolving by mechanical instrumentation which facilitates motility and thus also serves predatory purposes in acquiring food. Then there are the "trace" elements, essential to plant growth, an excess of which is toxic. In short, there is no such thing as a "linear" relation between life and the elemental forms of matter and energy. A threshold is the very essence of life.

Linear projections or extrapolations are a semantic fiction originating in the Euclidean concepts that arose in the days when the earth was called flat and everything in geometry was worked out in terms of rectilinear and rectangular frames of reference, a state of affairs that still plagues the problems of solid state physics. Molecular engineering, such as is characteristic of this atomic age,

cannot be conducted in these all too familiar terms. The microcosm, in short, is not at all a replica of the macrocosm, as it was held to be by no less a scientist than Thomas Huxley, father of the equally mistaken geneticist, Julian Huxley. This false analogy is being gradually abandoned though the layman is still deluded by the clutter of orbits in pictures of a now discarded concept of the behavior of electrons in an atom. "Shells" (energy "levels") have become the "truth"; and just now there seems a prospect that "spin" will also be displaced, perhaps by a helical structure. The whole theory of atomic and molecular models seems to be in a highly fluid state of uncertainty that is semantic rather than realistic in character. The meaning of symbols, such as the linear representations of Euclidean geometry, is at stake.

The truth about mathematical semantics seems to be that the so-called "natural" numbers, said to be an infinite continuum, are actually *un*natural in being a progression from an arbitrary origin (zero) in two "linear" directions. When they represent objective measurements of physical realities (rather than "value judgments" which can never be referred to any standard, subjective unit to give commutative character to their meaning) they are dimensional and, however remotely derived by instrumentation, they do form an integrated whole or system through reference to the ultimate unit, an arbitrary standard of length, to wit, the yardstick at the National Bureau of Standards. The resulting figures, called measurements, are always, whether "first" or last, approximations and never fully commensurable.

Truly *natural* numbers are dimension*less*; they have no quantitative meaning. Their field seem to be derived from the symmetries of microcosmic, spherical packing which emerge into specific forms in the periodic table of elements and in crystalline structures. Their order, if any, is timeless and independent of any comparison in size and they omit all but the smallest prime numbers since they are exponential and are not amenable to decimal treatment. No zero, no signs (plus or minus), no incommensurables, approximations or probabilities, no statistical averages, are involved in these symmetrical realities that never signify values either measured or subjectively appraised. Incidentally, it is not true that ratios are independent of dimensional numbers.

Assuredly, these venturesome generalizations need to be as critically reviewed as do the accepted conventionalities of mathematics. They are entered here heuristically, to suggest a clear distinction between certainty and uncertainty, a definitive difference between the possible and the impossible, in order to realize that the element of uncertainty is injected as soon as the information existing in the microcosm is "perceived," that is, *after* the optical structure of the eye, for example, has detected the signals and started, not merely to amplify, but to "comprehend" or classify them relative to the purposive procedures implicit in the neural system of any animal, such as a man or an insect. From then on there is a degree of uncertainty paralleling the logical doubt as to the truth of such semantic signs as are used in communication, even by honeybees.

There is nothing more irrational than the bland assumption that what is not yet known to exist must be considered not to exist, scientifically speaking. As Professor Ballard of Tulane University expresses this in the July, 1955, issue of Philosophy of Science, there is every reason to suspect that a dialectic comparable to human (and apian) language is "also carried on within an individual between distinguishable parts of his organism," a phenomenon which obviously presents "metaphysical" aspects and therefore "problems which mechanics cannot solve." Explanations which are a matter of inference by analogy, and not a matter of observation psychologically, thus lose their aura of mysticism and become scientifically anti-empirical. Rational strategy often rests, not on experimental verification of a suspicion (hypothesis) but on an "anthropomorphic" inference or belief that *all* behavior derives from communicated intelligence; subconscious action (instinctive) is not altogether mechanistic or free from awareness, not even in hereditary phenomena.

So meaning in communication is metaphysically incorporated and only approximately true at "best", while at "worst" (these antitheses are reversible in their "value" judgment, depending on whose ox is being gored) it is deliberately deceptive in its strategy, even though only by the "humor" of a pun. The living, purposeful organism has to learn not to be naive but to correct such illusions as that of the asymptotic approach in the perspective of distance or that of change in the pitch of a sound as its source moves past to create a "Doppler effect."

Dr. Schweitzer to the contrary notwithstanding, John Gunther's medical treatise on cancer and death is grounded on falsehood; for it is Life, not Death,

that needs to "Be Not Proud!" Immortality is impossible. Racial survival is possible, but not certain. Indeed, it too is impossible if all life—all mankind—is to be the objective. A Communistic Utopia is entirely outside the pale of any Creation except that of a demagogic imagination. Humility toward our competitors (our "fellow men") can only be hypocritical. Only the Creator (if any; we can never know), or better the actual order of the universe (which is not beneficent but is the very paradigm of neutrality) can be an object of respect and faith on the part of any moral culture seeking a political order not grounded on the personal discretion of leadership (der Feuhrerprinzip) in determining its blindfolded justice.

Occasionally the life of the individual can and must be treated with the utter *sang froid* of statistical probability and war, for it is always subordinate to the higher order that can be immortal. We can be legally exempt from taxes, but not from death. The termination of the life cycle can only be postponed, even by "Atoms in Our Future," the pleasant author of this pleasant prophesy, Senator Anderson, to the contrary notwithstanding. Men may be blessed with "travel out among the stars" and "pushbutton weather" (which is not altogether lacking even in New Mexico without benefit of atomic energy). But any promise of "even eternal life" is out of the reach of either man or God or, even, of the Positivism of such "science" as that which Sir George Thompson has set forth *ex cathedra* in his "The Foreseeable Future."

In her youth Lily Pons popularized "I Dream Too Much!" Perhaps her vibrant voice could yet teach it to Sir George and the prophetic Senator and persuade them and their sycophants in science to think in terms of thresholds rather than extrapolations.

Appendix 8

Radioactive Fallout

A Short Selected List of References

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APPENDIX 9

RADIOACTIVE FALLOUT

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Mice were exposed to atomic bomb radiation at 28 stations no both sides of the established L. D. $_{50}$ distance to compare the biological effect with that produced by laboratory gamma and X-radiations. At doses up to 620 r, mortality was consistently at 3 percent but above this dosage, mortality increased rapidly to 950 r, which was approximately the absolute lethal dose. The established L. D. $_{50}$ was 759 r. The relative biological effects of X-ray to bomb radiation closely approached unity.

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The basic mechanics of radioactive fallout are discussed. The principal isotopes are listed, collection stations are tabulated, and methods of analysis are briefly outlined. The isotope Sr^{90} is considered the constituent of prime biological significance.

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A network of 121 monitoring stations has been established in the United States to collect airborne and settled dust samples for radioactive assay. The results from 30,000 samples collected in conjunction with the 8 detonations in Nevada between April 1 and June 4, 1953, are given. For brief periods following an explosion the radioactive background can be increased in distant areas by fallout of airborne dust.

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Summarizes fallout in the United States from early in 1951 through 1954. Accumulated fission product activity in the United States, exclusive of the area within 200 miles of the Nevada test site, was 61 millicuries per square mile. Gamma radiation from this is of the order of 0.0010 mr/hr compared with normal background of 0.005 to 0.05 mr/hr. Measuring technique detects increase of 10^{-6} roentgens per hour, natural background 5×10^{-6} to 5×10^{-5} r/hr.

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This work was done to determine any radioactive increase following atomic weapons tests during 1955. Nine objectives are outlined. Analytical procedures are listed, and the results discussed. Materials of all kinds were

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Background measurements with a sodium-iodide scintillation detector at New York University show normal variation of ±5 percent. At 1030, March 19, 1953, the rate increased to 3850 cpm, 600 percent above background. A heavy rain preceded the measurement and presumably contained debris from the March 17 Nevada test. Removal of rainwater dropped rate to 1470 cpm. Curves of energy versus count before and after fallout are given.

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Radioactive concentrates were prepared from the atmosphere. Data of July 26 and August 30, 1946, represent the active deposits of Rn and Tn. The data of July 28, based on two samples with initial intensities of $\sim 5 \times 10^{-10}$ curie, are explained by assuming that the concentrate is the active deposit of a new rare radioactive gas of at. No. 86, with a halflife of 82 min.; it corresponds with at least two members of an unreported radioactive series.

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The isotopes of curium, berkelium, and californium found in the thermonuclear debris of the November 1952 thermonuclear test are discussed. The instantaneous buildup of the heavy elements in the thermonuclear device is compared with the buildup during pile irradiation. The alpha-particle energy (5.4 Mev) and the spontaneous fission half-life ($<1.2 \times 10^7$ years) of Cm^{240} are reported. The spontaneous fission halflife of Cf^{254} was found to be 55 days. No other mode of decay was observed for this isotope.

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Issue contains an editorial on genetics in Geneva, glossary of genetic terms and articles on the subject.

Greenfield, S. M. Ionization of radioactive particles in the free air. Journal of geophysical research, v. 61, 1956: 27–33.

In order to evaluate the possible role of radioactive particles from an atomic cloud as condensation nuclei, an analysis has been made to determine their degree of ionization. Individual radioactive particles become ionized owing to β-emission, and an estimate of the half-life of these ions has been made for various times in the life history of an atomic cloud. It is concluded that while there is a transient charge on these particles, its half-life is small compared to the disintegration rate, with the result that for all practical purposes radioactive particles in the free air are not necessarily preferred condensation nuclei.

——— Rain scavenging of radioactive particulate matter from the atmosphere. Journal of meteorology, v. 14, April 1957: 115–125.

Hahn, Richard B. and Straub, Conrad P. Determination of radioactive strontium and barium in water. Journal of American Water Works Association, v. 47, April 1955: 335–340.

Haldane, J. B. S. Genetical effects of radiation from products of nuclear explosions. Nature, (London) v. 176, July 16, 1955: 115.

The serious nature of genetical effects of radiation is argued. Upper and lower limits of radiation-induced human mortality are estimated.

Harris, D. Lee. Effects of atomic explosions on the frequency of tornadoes in the United States. Monthly weather review, v. 82, December 1954: 360–369.

The increase in tornadoes reported in the United States during the past few years is ascribed to better reporting procedures rather than the presence of atomic debris. Maps showing the distribution of fallout in the United States during the first and second halves of May 1953 are given.

Harris, William B., and LeVine, Harris D. Sampling and measurement of radioactive atmospheric pollution. Proceedings of the Air Pollution Control Association, 1953: 17–21.

Apparatus is described for continuously monitoring the alpha and gamma radiation from stack effluents. A storage-battery-driven dust collector can be used to collect dust away from power sources. An adhesive-coated film can be used to collect "fallout," at a distance from the source. An apparatus is shown for collecting samples nearer the source of contamination by the use of impactors, cyclones, etc.

Harris, D. Lee. Effects of radioactive debris from nuclear explosions on electrical conductivity of lower atmosphere. Journal of geophysical research, v. 60, March 1955: 45–52.

An increase in the ionization near the ground due to the fallout from a radioactive cloud formed by a nuclear explosion will increase the conductivity and lower the potential gradient in the lower atmosphere. Records of atmospheric conductivity and potential gradient from the Tucson Magnetic Observatory are compared with records of the deposition of atomic debris on the ground following the Nevada tests.

Herzog, G. Gamma-ray anomaly following the atomic bomb test of July 1, 1946. Physical review, v. 70, 1946: 227–228.

A recording gamma-ray meter (in Houston, Tex.) indicated a peak in atmospheric gamma rays, from 8 p. m., July 4 to 7 p. m., July 5, with a maximum at 3 a. m., July 5. The maximum increase was 77 percent of the background count.

Heslep, J. M., and Bellamy, A. W. Sampling for airborne radioactivity. Air repair, v. 5, May 1955: 1–4.

Potential sources of radioactive aerosols are discussed, and special attention is given to widespread contamination as might be expected from atomic weapons. There is no perfect sampling method, but rather good results have been secured by use of con. vacuum cleaners and Hollingsworth and Vose H–70 filter paper. Particle-size determinations are best made by the casca de impactor—this gives good characterization of particle size down to 0.3–0.6 μ

Hess, Victor F. and Luger, Paul. The ionization of the atmosphere in the New York area before and after the Bikini atom-bomb test. Physical review, v. 70, 1946; 564–565.

From June 29 through July 10, 1946, no atmospheric ionization due to the atomic bomb was observed.

Hollaender, Alexander. Modification of radiation response. Bulletin of the atomic scientists, v. 12. March 1956: 76–80.

Holzman, B. The effects of atomic bomb explosions on weather. Weatherwise, v. 4, February 1951: 3–4 f.

Holter, N. J. and Glasscock, W. R. Tracing nuclear explosions. Nucleonics, v. 10, August 1952: 10–13.

Airborne radioactivity precipitated in rain and snow has been measured by counter observations on samples concentrated by evaporation or filtration through cotton. A maximum half life of 10.6 hours (Pb^{212}) is associated with natural atomspheric activity. A number of samples collected (at Helena, Mont.) revealed activities of much longer decay periods. These are attributed to atomic explosions in Nevada and Russia. It is considered possible to assign a date to the occurrence of the explosion from observations of the decay curve.

Humphrey, Andrew J. Radiation injury: a technical and legal survey. Cleveland—Marshall law review, v. 6, May 1957: 171–188.

Examines the relations between various types of radiation as to sources and effects.

Hunter, H. F. and Ballou, N. E. Fission-product decay rates. Nucleonics, v. 9, November 1951: C–2–C7.

Jaffee, Gilbert, and others. Radioactive hailstones in the District of Columbia, May 26, 1953. Bulletin of the American Meteorological Society, v. 35, June 1954: 245–249.

At 2030 Greenwich civil time on May 26, 1953, hailstones ranging to the size of tennis balls fell in the District of Columbia, 29 hours after an atomic test in Nevada. Activity of 620 c/m as compared to a background of 20 c/m was measured in the stones. Meteorological analysis and decay curves confirmed the origin of the radioactivity as being from the atomic test.

Kellogg, D. A. Atomic defense in oil refinery. Petroleum engineering, v. 27, October 1955: C6–C8.

Methods of preventing continuous effects of radioactive fallout.

Kellogg, W. W., and others. Close-in fallout. Journal of meteorology, v. 14, February 1957: 1–8.

Keosian, John. Speculation on hazards of exposure to radiations. Science, v. 122, September 30, 1955: 586–587.

Question of maximum tolerance dose of radiation for man has not been satisfactorily determined. It may turn out that all high energy radiation, even of low intensity and brief duration must be considered as potentially dangerous to the exposed individual.

Kilcawley, E. J. Measurement of radioactive fallout in reservoirs. Journal of the American Water Works Association, v. 46, November 1954: 1101–1111.

An extensive survey of radioactivity in the Troy, Albany, Schenectady water supply system was made in an investigation following the heavy rainout which occurred in that area on April 26, 1953. Samples of water, soil, algae, plants, etc., were measured for radioactivity. Decay rates, effectiveness of filtration, rate of disappearance, etc., were studied. The general contamination of the ground at arrival time was 1μc/ft^2. Surface water at Rensselaer Polytechnic Institute, Troy, N. Y., had 2.7 x 10^{-2} $\mu\mu$c/ml. Measurements were also made for subsequent bursts. Corrected to time of rainout, highest rainwater activity was 25.0 $\mu\mu$c/ml on June 9, 1953. Highest stream samples, 0.10–.13 $\mu\mu$c/ml. Alpha activity of same samples was also investigated.

Kirby-Smith, J. S. and Swanson, C. P. The effects of fast neutrons from a nuclear detonation on chromosome breakage in tradescantia. Science, v. 119, January 1, 1954: 42–46.

Physical determination of the fast neutron dose in nuclear explosions; supplementary experiments to those of Conger.

Krumholz, Louis A. Observations of the fish population of a lake contaminated by radioactive wastes. Bulletin of the American Museum of Natural History, v. 110, article 4, 1956: 281–367.

Kulp, J. Laurence, and others. Strontium-90 in man. Science, v. 125, February 8, 1957: 219–225.

To determine amount of radioactive strontium in human bones today, three scientists from Columbia University analyzed about 500 autopsy samples obtained from 17 stations in a worldwide network. Concludes that if bomb tests continue at their present rate the average worldwide concentration in 1970 will be 4 to 8 micromicrocuries of strontium-90 per gram of calcium. The upper figure approaches the significant level established by the British Medical Council in its June 1956 report.

Lacassagne, A. The risks of cancer formation by radiations. Bulletin of the atomic scientists, v. 13, April 1957: 135–136f.

Langham, Wright H., and Anderson, F. C. Strontium-90 and skeletal formation. Science, v. 126, August 2, 1957: 205–206.

Lapp, Ralph E. Strontium-90 in man. Science, v. 125, May 10, 1957: 933–934.

Commentary on Kulp article in Science, February 8, 1957.

Reply by Kulp and others: 934.

Lewis, E. B. Leukemia and ionizing radiation. Science, v. 125, May 17, 1957: 965–972.

Discusses incidence of leukemia in Hiroshima and Nagasaki, also cases among radiologists. Applied to radiostrontium exposures.

Libby, Willard F. Current research findings on radioactive fallout. Proceedings of the National Academy of Sciences, v. 42, December 1956: 945–962.

Speech given before American Association for the Advancement of Science, October 12, 1956.

———. Dosages from natural radioactivity and cosmic rays. Science, v. 122, July 8, 1955: 57–58.

Reprinted in Congressional Record (Daily ed.) July 14, 1955: A5165–A5166.

———. Genetic effects of atom bombs. Metal progress, v. 68, October 1955: 130–131.

Libby, Willard F. Radioactive strontium fallout. Proceedings of the National Academy of Sciences, v. 42, June 1956: 365–390.

Based on a speech given before Annual General Meeting of the American Philosophical Society, April 20, 1956.

Hazards from Sr^{90} deposited in fallout following nuclear explosions are reviewed. Strontium^{90} is of particular interest among the fission products because of chemical similarity to Ca, an average life of about 40 years, and a low rate of skeletal elimination. The maximum permissible average concentration of Sr^{90} in the adult skeleton is calculated to be 1μc/1,000 gm of Ca.

Data are summarized on Ca, Sr, and Sr^{90} concentration in samples of soil, animal, and plant material collected throughout the world before and after the thermonuclear explosions during Operation Castle. A Sr^{90} fallout probably derived from megaton weapons are nearly uniform over the world except for local effects due to rainfall variations and to fallout from submegaton weapons, was found to occur at least 1.7 years after the megaton test series. The average world-wide Sr^{90} fallout rate in the fall of 1954 and the spring and summer of 1955 was 1.2 mc/mi^2/yr. An estimate is presented of fallout rate of Sr^{90} to be expected from weapons tests up to and including the Castle series. Factors influencing the transfer of Sr^{90} from soil to plants, to animals and milk produced by them, and finally to the human skeleton are discussed.

Lieberman, Joseph A. Disposal of radioactive wastes—a growing problem. Civil engineering, v. 25, July 1955: 44–47.

List, Robert J. On the transport of atomic debris in the atmosphere. Bulletin of American Meteorological Society, v. 35, September 1954: 315–325.

Describes results of 91 gummed paper and 51 air filter monitoring stations in the United States during Nevada tests in the spring of 1952. Detailed meteorological trajectories of each of the bursts of the series are given, together with a discussion of the meteorological aspects of transport and fallout. Excluding area within 200 miles of test site, highest gummed paper fission product beta activity was 8×10^6 d/m/ft^2/day at station 330 miles from test side. At distances over 2,000 miles, maximum activity was 1.7×10^5 d/m/ft^2/day, on sampling day. The two highest air filter activities were 1.3×10^5 and 6.8×10^3 d/m/meter^3. Detailed discussion of fallout from 3 of the 8 bursts given, with daily maps showing isolines of activity and areas of precipitation for several days following the bursts.

———. On the transport of atomic debris in the atmosphere. Journal of the Air Pollution Control Association, v. 5, 1955: 153–156f.

The author correlated the meteorological trajectories of bomb debris following each of the eight nuclear detonations at the Nevada test site in 1952 with fallout in the United States. In most instances the predicted pattern of fallout was in agreement with fallout data.

Luntz, Jerome D. Radiation safety for a weapons test. Nucleonics, v. 10, May 1952: 10–13.

An eyewitness report on the elaborate system used to obtain detailed data on distribution of radioactivity from the atomic explosion on April 22, 1952.

Machta, L. and Harris, D. L. Effects of atomic explosions on weather. Science, v. 121, January 21, 1955: 75–81.

A study of temperature and rainfall for the United States does not indicate any departures from normal that are related to atomic explosions.

Machta, L., and others. Worldwide travel of atomic debris. Science, v. 124, September 14, 1956: 474–477.

Machta, L., and others. Airborne measurements of atomic debris. Journal of meteorology, v. 14, April 1957: 165–175.

Margolis, Emanuel. The hydrogen bomb experiments and international law. Yale law journal, v. 64, April 1955: 629–647.

Detailed consideration of influence of such international law doctrines as freedom of the seas and the illegality of the "pollution" of international waters on the H-bomb test in the Pacific.

McDougal, M. S. Hydrogen bomb tests and the international law of the sea. American journal of international law, v. 49, July 1955: 356.

——— and Schlei, N. A. Hydrogen bomb tests in perspective: lawful measures for security. Yale law journal, v. 64, April 1955: 648–710.

Discusses conflicting claims of the security of the United States and its allies and the principles of international law as interpreted in some quarters.

Meinke, W. Wayne. Observations on radioactive snows at Ann Arbor, Mich. Science, v. 113, May 11, 1951: 545–546.

Rigorous chemical separations performed on radioactivities found in snows around Ann Arbor, Mich., after the Las Vegas atomic test explosions on January 27 to February 6, 1951, have definitely established the presence of radioactive rare-earth isotopes and Ba and/or Sr isotopes and have shown the possible presence of I isotopes. The tests conducted on the samples are described. Because of the chemical distribution of the activities found in the Ann Arbor snows, these activities undoubtedly originated in the Las Vegas atomic test explosions.

Mesler, Russell B. and Widdoes, Lawrence C. Evaluating reactor hazards from airborne fission products. Nucleonics, v. 12, September 1954: 39–41.

Meteorological aspects of atomic radiation. Science, v. 124, July 20, 1956: 105–112.

One of six reports prepared for the Study of the Biological Effects of Atomic Radiation by the National Academy of Sciences.

Miller, C. E. and Marinelli, L. D. Gamma-ray activity of contemporary man. Science, v. 124, July 20, 1956: 122–123.

Moloney, William C. and Kastenbaum, Marvin A. Leukemogenic effects of ionizing radiation on atomic bomb survivors in Hiroshima City. Science, v. 121, February 25, 1955: 308–309.

Incidence of leukemia is "high" at distances close to the hypocenter, regardless of presence or absence of severe radiation complaints.

Morgan, K. Z. Maximum permissible internal dose of radionuclides: recent changes in values. Nuclear science and engineering, v. 1, December 1956: 477–500.

Muller, Hermann J. After effects of nuclear radiation. National safety news, v. 74, August 1956: 43–48. Bibliography.

———. Genetic damage produced by radiation. Science, v. 121, June 17, 1955: 837–840.

———. The genetic damage produced by radiation. Bulletin of the atomic scientists, v. 11, June 1955: 210 ff.

Article based upon Japanese analyses of fallout from the March 1, 1954, superbomb.

———. How radiation changes the genetic constitution. Bulletin of the atomic scientists, v. 11, November 1955: 329–338.

———. Radiation and human mutation. Scientific American, v. 193, November 1955: 58–68.

———. Radiation damage to genetic material, Parts I and II. American scientists, v. 38, 1950: 33, 399.

Nader, J. S., and others. Radioactive fallout in rain in the Cincinnati area. Journal of the American Water Works Association, v. 46, November 1954: 1096–1100.

Precipitation in the Cincinnati area was measured for suspended and soluble radioactivity from March 1953 to March 1954. "Background" level in precipitation was 0.03 to 0.08 μμc/ml. Maximum concentration, on April 29, 1953, 319 μμc/ml (500 μμc/ml corrected for decay). Maximum fallout from a single rain on May 22, 1953, 1.55 inches containing 85.7 μμc/ml, giving 8.75 curies per square mile. Soluble activity averaged 60–80 percent of total before and after tests, fell to 30 percent during tests. Samples from creeks and tapwater show most of activity removed by natural purification. Accumulated and decayed rain activity on June 10 and December 10, 1953, was 2.4 and 0.15 curies per square mile, respectively.

A Navy medical team studies fallout effects. Bulletin of the atomic scientists, v. 12, February 1956: 58–59.

Reprint of article on radiation research performed by doctors from Naval Medical Research Institute after 1954 nuclear tests in Marshalls. Original article in Research Reviews, November 1955. Also summarized in Science, v. 122, December 16, 1955: 1178–1179.

Neher, H. V. Gamma rays from local radioactive sources. Science, v. 125, May 31, 1957: 1088–1089.

New research facts on how foods weather A-bombing. Food engineering, v. 28, November 1956: 59f.

Oceanography, fisheries and atomic radiation. Science, v. 124, July 6, 1956: 13–16.

Part of a continuing study on the Biological Effects of Atomic Radiation conducted by the National Academy of Sciences.

Ophel, I. L. Fallout and the strontium-90 hazard. Science, v. 125, March 1, 1957: 399.

Plough, H. H. Radiation tolerance and genetic effects. Nucleonics, v. 10, 1952: 16–20.

Radiation and health (editorial). Science, v. 125, April 19, 1957: 719.
Questions necessity for establishing a National Radiation Health Institute in the Public Health Service. Points out that any radiation health agency should deal with radiation from all sources, not just atomic radiation.

Randolph, M. L., and others. Effect of bone-marrow treatment on mortality of mice irradiated with fast neutrons. Science, v. 125, May 31, 1957: 1083–1084.

Rediske, J. H. and Selders, A. A. The absorption and translocation of strontium by plants. Plant physiology, v. 28, 1953: 594–605.

Rosenfeld, A. H., and others. Fallout: some measurements and damage estimates. Bulletin of the atomic scientists, v. 11, June 1955: 213–216.

Rotblat, Joseph. The hydrogen-uranium bomb. Bulletin of the atomic scientists, v. 11, May 1955: 171–172, f.
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Russell, W. L. Comparison of X-ray induced mutation rates in drosophila and mice. American naturalist, v. 90, January–February 1956: 69–80.
Until recent years, estimates of genetic hazards of ionizing radiation in man were based primarily on information obtained from drosophila. Investigations on radiation-induced mutation rates in mice showed a higher mean rate in the mouse and led to conclusion that estimates of human hazards based on drosophila mutation rates may be too low.

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———. Shortening of life in the offspring of male mice exposed to neutron radiation from an atomic bomb. Proceedings of the National Academy of Sciences, v. 43, Apr. 1957: 324–329.

Schubert, Jack. Radioactive poisons. Scientific American, v. 193, August 1955: 35–39.
The biological effects of nuclear radiation are as yet imperfectly understood. Maximum permissible limits of exposure must be determined more precisely if byproducts of nuclear technology are to be safely handled.

Setter, L. R., and Goldin, A. S. Radioactive fallout in surface waters. Industrial and engineering chemistry, v. 48, February 1956: 251–255.

Slatis, Herman M. Current status of information on the induction of mutations by irradiation. Science, v. 121, June 10, 1955: 817–821.

Some biological effects of radiation from nuclear detonations. American naturalist, v. 88, 1954: 209–314.

Stefannizzi, A. Radioactivity of atmospheric precipitates. Journal of geophysical research, v. 55, 1950: 373–378.
The radioactivity of snow and rain was detected in 33 cases. It was found that snow usually has a greater activity than rain; that rain in thundershowers is more active than ordinary rain; and that at least a certain amount of activity is acquired by precipitates during their fall from the clouds to the ground level.

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Sturtevant, A. H. The genetic effects of high energy irradiation of human population. Engineering and science, v. 18, January 1955: 9–12.

———. Social implications of the genetics of man. Science, v. 120, September 10, 1954: 405–407.

Tajima, Eizo and Doke, Tadayoshi. Airborne radioactivity. Science, v. 123, February 10, 1956: 211–214.
Twenty-four hour air filters in Tokyo from March 16 to May 31, 1955, were measured for artificial and natural radioactivity. The samples consisted of 650 cubic meters per day and assuming a 10 percent collection efficiency of the Whatman No. 14 filter papers for small particles, the peak activity from fission products, observed on April 12, was $3 \times 10^{-15} \sim 1.2 \times 10^{-14}$ curies per liter, which is comparable to the concentration of natural activity. No consistent correlation of fission product activity with temperature was found and only a slight tendency for rainfall to clean the air was noted. A better correlation was observed with trajectories of high pressure areas.

Thomas, Harold A., and others. Radioactive fallout in Massachusetts surface waters. Journal of the American Water Works Association, v. 45, June 1953: 562–568.
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Thompson, Raymond. They play tag with atomic clouds. Science digest, v. 38, August 1955: 9–12.
Work of planes and crews from Air Force's Air Research and Development Command in tracking radioactive clouds after test atomic explosions.
Toombs, Alfred. Radioactive "garbage"—newest threat to man and nature. Natural history, v. 65, September 1955: 344–349.
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Van Middlesworth, L. Radioactivity in thyroid glands following nuclear weapons tests. Science, v. 123, June 1, 1956: 982–983.
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Warren, Shields. Antipersonnel effects of nuclear weapons. Confluence, v. 5, July 1956: 131–138.
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Symposium paper presented at all-day scientific program on the uses and effects of atomic radiation held by the American Association for the Advancement of Science, October 12, 1956 at the Carnegie Institution of Washington.
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Wright, J. H., and others. High-speed computer for predicting radioactive fallout. Journal of research of the National Bureau of Standards v. 58, February 1957: 101–109.
Yoshii, G., and others. Biological decontamination of fission products. Science, v. 124, August 17, 1956: 320–321.

MEDICAL JOURNAL ARTICLES

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Bergsma, Daniel. Public health aspects of atomic energy in peacetime. Public health reports, v. 71, January 1956: 43–46.
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Presented before the Radiation Research Society, New York, May 16–18, 1955.

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Cronkite, Eugene P. Radiation injuries of atomic warfare; pathogenesis and therapy. Journal of the Omaha Midwest Clinical Society, v. 13, January 1952: 6–13.

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Glass, Bentley. The hazards of atomic radiations to man. Journal of heredity, v. 47, 1956: 260.

Greulich, W. W., and others. Physical growth and development of children who survived bombing of Hiroshima or Nagasaki. Journal of pediatrics, v. 43, August 1953: 121–145.

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Muller, Hermann J. Comments on the genetic effects of radiation on human population. Journal of heredity, v. 46, 1955: 199–200.

———. Damage to posterity caused by the irradiation of the gonads. American journal of obstetrics and gynecology, v. 67, 1954: 467–483.

Powell, Clinton C. Medical aspects of the control of radiologic health problems. American journal of public health, v. 47, February 1957: 179–183.

Reynolds, Mardelle L. and Lynch, Francis X. Atomic bomb injuries among survivors in Hiroshima. Public health reports, v. 70, March 1955: 261–270.

Rolleston, H. Critical review of the harmful effects of irradiation (X-rays and radium). Quarterly journal of medicine, v. 23, 1930: 101–132.

Russell, L. B. and Russell, W. L. Radiation hazards to the embryo and fetus. Radiology, v. 58, 1952: 369–376.

Salvin, M. Effect of atomic radiation on the incidence of sterility and mutation. Journal of the National Medical Association, v. 48, March 1956: 79–83.

Stoll, B. A. New drugs for irradiation sickness. Journal of radiology, v. 68, March 1957: 380–385.

Stone, R. S. The concept of a maximum permissible exposure. Radiology, v. 58, 1952: 639–661.

Storer, J. B., and others. Relative biological effectiveness of various ionizing radiations in mammalian systems. Radiation research, v. 6, February 1957: 188–288.

Straub, C. P. Limitations of water treatment methods for removing radioactive contaminants. Public health reports, v. 70, September 1955: 897–904.

Sutow, Wataru W., and West, Emory. Studies on Nagasaki (Japan) children exposed in utero to the atomic bomb. A roentgenographic survey of the skeletal system. American journal of roentgenology, radium therapy and nuclear medicine, v. 74, September 1955: 493–499.

Taliaferro, William H. and Taliaferro, Lucy G. Effect of X-rays on immunity: a review. Journal of immunology, v. 66, February 1951: 181–212.
Review documented by 201 references.

Taylor, Lauriston S. Education in radiation protection. American journal of roentgenology, radium therapy and nuclear medicine, v. 73, February 1955: 193–202.

———. Legislative control of radiation. Radiology, v. 66, February 1956: 246–252.
"Two States already have radiation-control regulations, and several others are in the process of developing them * * * The National Committee on Radiation Protection feels that legislation should cover both occupational and non-occupational exposure and all kinds of radiation * * * The Committee believes that the maintenance of the maximum degree of uniformity between the regulations in all States is of prime importance."

———. State control of protection against ionizing radiation. American journal of roentgenology, radium therapy and nuclear medicine, v. 71, April 1954: 691–702.

Thomas, Harold A. The public health implications of radio-active fallout in water supplies. American journal of public health, v. 46, October 1956: 1266–1274.

Warren, Shields. Public health aspects of atomic power development. A. M. A. Archives of industrial hygiene and occupational medicine, v. 9, March 1954: 183–185.

———. Longevity and causes of death from irradiation in physicians. Journal of the American Medical Association, v. 162, September 29, 1956: 464–468.

Williams, E. G., and Ingraham, S. C. Biological-medical considerations in atomic defense. Public health reports, v. 71, February 1956: 173–180.

Wilson, Robert R. Nuclear radiation at Hiroshima and Nagasaki. Radiation research, v. 4, 1956: 349–359.

Use of X-rays and other ionizing radiation; U. N. Scientific Committee on Effects of Atomic Radiation. Armed Forces medical journal, v. 8, March 1957: 358–360.

Vorder Bruegge, C. F. Radiation injury following A-bomb explosion. Annals of internal medicine, v. 36, June 1952; 1444–1458.

Ward, L. J. Lethal radiation from exploded atomic bomb. Mississippi Valley medical journal, v. 74, November 1952: 191–195.

Yamazaki, J. N., and others. Symposium on effects of radiation and other deleterious agents on embryonic development; study of outcome of pregnancy in women exposed to atomic bomb blast in Nagasaki. Journal of cellular and comparative physiology (supp. 1) v. 43, May 1954: 319–328.

Zavon, M. R. Radiation—helpful or harmful? Journal of the American Medical Association, v. 162, 1956: 533.

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BRITISH

Auerbach, Charlotte. Biological hazards of nuclear and other radiations. Nature (London) v. 178, September 1, 1956: 453–454.
A comparison is made of British and United States reports on radiation hazards. Both reports show that the present dangers arise much more from excessive use of X-rays than from fallout or atomic energy establishments.

Biological effects of radiation. Nature (London) v. 179, April 13, 1957: 755–756.

Blifford, Irving H., and others. Relation between air concentration of radioactive fission products and fallout. Nature (London) v. 177, 1956: 990–992.
The daily atmospheric concentration and fallout of radioactivity due to fission products was measured at Washington, D. C., during December 1954–May 1955 by air-filter and gummed-paper techniques, respectively. The apparent rate of decay was used to distinguish between natural Th B and fission products. There was no correlation of individual daily measurements of air concentration and fallout of this material on the ground. Despite many variables the concept of fallout rate may be useful in arriving at some correlation.

Chatterjee, Santimay. Radioactive ashes over Calcutta and a method of dating a nuclear explosion. Atomic scientists journal (London) v. 4, 1955: 273–278.

Cockcroft, John D. Biological effect of nuclear explosions. Pharmaceutical journal (London) v. 174, 1955: 387–388 f.

———. Radiological hazards from nuclear explosions and nuclear power. Nature (London) v. 175, May 21, 1955: 873–875.

Reprint of address to the Parliamentary and Scientific Committee at the House of Commons, London, April 20, 1955.

Average concentration of radioactivity in the air at ground level over the last 3 years due to bomb explosions is about 1 percent of the natural radioactive dust content. Accumulated dose to completely unprotected people from fallout in England is about 0.01 R. Further fallout from airborne debris should bring it to 0.03 R. In the United States, average dose is about 0.1 R. Average dose in England to average person over a generation (30 years) will be about 0.003 R, natural radioactivity gives 3 R. Discusses other radiological hazards and concludes that probably at least 1,000 times the present level of contamination would be needed to give rise to serious harmful effects.

Howard, Alma. The hazards from the increasing use of ionizing radiations: A symposium. III. An attempt to assess the genetic changes resulting from the irradiation of human populations. British journal of radiology, (London) v. 29, June 1956: 270–273.

Leukaemia and natural background radiation. British medical journal, issue 5021, March 30, 1957: 760.

Nishiwaki, Yasushi. Bikini ash. (Letter to the editor.) Atomic scientists journal (London) v. 4, November 1954: 97–109.

Account of the results of the shower of radioactive ash that followed the explosion of the H-bomb at Bikini. Effects of the fallout on the Japanese fishermen, on the fish in the sea, etc., are considered.

———. Effects of H-bomb tests in 1954. Atomic scientists journal (London) v. 4, May 1955: 279–288.

Preston, R. L. and Hogg, B. G. Radioactive fallout in Kingston, Canada. Nature (London), v. 176, 1955: 459.

The monitoring of the radioactive fallout in Kingston, Ontario, during the period February 15 to May 28, 1955, from the nuclear tests conducted by the AEC is reported. Meteorological factors seem to outweigh the magnitude of the bombs at such a great distance from the explosion.

Poyce, M. H. L. Global thermonuclear explosions are impossible. Discovery (Norwich, England) v. 16, December 1955: 495–497.

Some people are afraid that a thermonuclear explosion might set off a reaction in the earth's crust and in the seas, resulting in the earth's destruction. The author of this article attempts to show why this is not possible.

Radiation hazards. Lancet (London), v. 270, June 23, 1956: 999–1000.

Radiation hazards of experimental nuclear explosions. British medical journal (London) No. 4916, March 26, 1955: 775–776.

Read, John. The approach of the physicist to radiation biology. Physics in medicine and biology (London) v. 1, January 1957: 209–224.

Rotblat, Joseph. The hydrogen-uranium bomb. Atomic scientists journal (London) v. 4, March 1955: 224–228.

This British scientist feels that the various bombs that have been detonated throughout the world have resulted in more dangers from radioactivity than officials admit. He believes that genetic aberrations may well result from present radioactivity in the atmosphere.

Sevitt, S. The bombs. Lancet, v. 269, July 23, 1955: 199–201.

———. The case of Mr. Kuboyama; a clinical and pathological study of the first victim of the atomic bomb. Medical world (London), v. 84, May 1956: 385–390.

Spiers, F. W. Radioactivity in man and his environment. British journal of radiology (London), v. 29, August 1956: 409–417.

———. and Haldane, J. B. Genetical effects of radiation from products of nuclear explosions. Nature (London) v. 177, February 4, 1956: 226–227.

Relative radiation dose-rates to man and to Drosophila are discussed. Data previously presented by Prof. J. B. S. Haldane on the genetical effects of radiation resulting from nuclear explosions are reviewed. A reply from Professor Haldane presents revised calculations of radiation dose rates.

Stanford, R. W., and Vance, J. The quantity of radiation received by the reproductive organs of patients during routine diagnostic X-ray examinations. British journal of radiology, v. 28, May 1955: 266–273.

Strontium-90 in man. (Letters to the editor.) British medical journal, issue 5024, April 20, 1957: 943–944.

Strontium-90 in man. British medical journal issue 5021, March 30, 1957: 752–753.

Sutton, G. Thermonuclear explosions and the weather. Nature (London) v. 175, February 19, 1955: 319–321.

Available evidence points to conclusion that atomic tests cannot be held responsible for any worldwide extremes of weather encountered in 1954.

FRENCH

Abribat, Marcel and Pouradier, Jacques. Evolution of the amount of artificial radioactive elements in the atmosphere of Paris. Comptes rendus, v. 237, 1953: 1233–1255 (in French).

The β-activities of airborne dust particles, rain waters, and natural waters were assayed daily between January and September 1953. The dust-particle activity showed 2 maximum March 24–June 25 and August 30–September 17, which probably resulted from atomic explosions in the United States and Russia, respectively. Rainwater activity gave only the earlier maximum while no changes in the activity of natural waters were found.

———, and others. Artificial radioactivity in rainwater of the Paris area. Comptes rendus v. 234, March 10, 1952: 1161–1163 (in French).

Considerable radioactivity was observed 8–15 days after the explosion of atomic bombs in Nevada.

——— ———. Evolution of atmospheric radioactivity in Paris region. Comptes rendus v. 240, 1953: 2310–2312 (in French).

Daily measurements of radioactivity have shown the passage of many atomic clouds, and particularly the series of explosions in the United States of America and Russia, while those in the Pacific and Australia have been identified in Milan. For the Australian explosion in October 1953 there was no radioactive increase in the air in the Paris region, while for the Pacific explosion there were measurable fluctuations but very feeble. For the Russian explosions in August 1954 the fluctuations were much greater than for the Pacific ones.

——— and others. On the artificial radioactive products present in the atmosphere in the region of Paris. Comptes rendus hebdomadaires des séances de l'Académie des Sciences (Paris) v. 235, July 16, 1952: 157–159(in French).

The radioactivity of solid matter in rain water and air collected near Paris in November 1951, and in April and May 1952, follows the same decay law as that observed for fission products after a nuclear detonation in Nevada in November 1951.

Besson, A. and Pelletier, J. Radioactivity and air pollutions. Bulletin de L'Académie Nationale de Médecine (Paris), par. 140, January 24–31, 1956: 43–45.

Bouquiaux, J. Ionizing radiations and public health problems. Archives Belges de médecine sociale, hygiéne, médecine du travail et médecine légale (Brussels). v. 14, May 1956: 230–268 (in French).

Garrigue, Hubert. The abnormal radioactivity of the atmosphere. Comptes rendus, v. 235, 1952: 1489–1499 (in French).

Further measurements on the radioactivity of the atmosphere near Puy de Dome are tabulated. The activity in April 1952 with a mean life of 25 hours was ascribed to the complex particles A. In June 1952 an activity with a mean life of 100–400 hours was attributed to the previously observed A″ particles. The activity associated with the A″ particles was also observed in the soil and air.

———. Atmospheric radioactivity of atomic origin. Comptes rendus, v. 237, November 16, 1953: 1232.

Aircraft measurements in October 1953, show at most, traces of radioactivity. Snow sample (7 kg) on November 3, 1953, yielded 0.1×10^{-13} curies/cm ^{3} (radon equivalent) in the residue, about 20 percent above background. Only β—activity was found.

———. Atmospheric radioactivity of atomic origin. Comptes rendus, v. 240, January 10, 1955: 178–180 (in French).

An improved collector has been installed in the aerial laboratory, but in recent months little activity caused by atomic explosions has been detected.

Garrigue, Hubert. Establishment of a flying laboratory and the improvement of apparatus for the study of weak radioactivity in the atmosphere. Comptes rendus, v. 230, June 26, 1950: 2279–2280 (in French).

Describes in general terms the equipment of a plane to measure the radioactivity of the air. The first flight, on May 13, 1950, showed a high concentration of natural activity and a measurable amount of activity of unknown origin with a long half-life.

———. Fresh outbreak of activity of atomic origin in the atmosphere. Comptes rendus, v. 240, 1955: 1453–1455 (in French).

During the period December 1954 to February 1955 values of the order of 1 β-emitting atom per cm^3 of air have been obtained for observations on the ground and in the air in Puy de Dôme region.

———. The invasion of radioactive air of atomic origin and its influence on atmospheric precipitation. Comptes rendus, v. 232, March 5, 1951: 1003–1004 (in French).

Reports on aircraft observations in June 1950 and in January and February 1951. On February 3, 1951, at 4,100 meters, a maximum concentration of 0.15×10^{-15} curies/cm^3 radon equivalent of material of 30–50 hour apparent half-life. Attempts to show that precipitation cleanses the tropospheric air.

———. Observations on the impurities in free air. Comptes rendus, v. 236, 1953: 2309–2311 (in French).

Results of analysis of atmospheric radioactivity and pollution from measurements on free air and on atomic precipitation (snow) at heights from ground level to the summit of the Puy de Dôme (1450 m) during January-February 1953. The radioactivity was probably of distant origin (from nuclear fission explosions); the dust and soot of local origin.

———. On the radioactivity of the atmosphere. Comptes rendus, v. 228, May 16, 1949: 1583–1584 (in French).

A radioactive substance with a half-life of 25 hours was detected in aircraft flights at about 6,000 meters in the summer of 1946 and again, in much weaker concentration, in the summer of 1948. Highest value reported, August 1, 1946, at 6,000 meters, 2.0×10^{-15} curies/cm^3. Observations at Puy-de-Dôme, 1,500 meters, gave no definite positive results. Speculation that material is from Bikini tests or possibly of meteoric or cosmic ray origin.

———. Prospecting the radioactivity of the air. Comptes rendus, v. 237, October 12, 1953: 802–803 (in French).

Measurements aboard an airplane (3,000 meters altitude) indicated a sudden influx of radioactive particles "A" with a mean life of 25 hours on August 8 (traces) and, in appreciable amounts, on August 15, 28, and September 5, 1953. The maximum of intensity of about 0.1×10^{-15} equivalent curie of Rn activity/cc. probably prevailed around August 15. No activity could be detected prior to these dates in air or in snow on the summit of Puy-de-Dôme (1,460 meters), nor did the evaporation of hail (collected on the ground), precipitated during a "microcyclone," which occurred on August 8, disclose any abnormal activity.

———. Radioactivity of the atmosphere of atomic origin. Comptes rendus, v. 237, 1953: 1232–1233.

Recent measurements at the summit of Puy-de-Dôme indicate the radioactivity of the snow may be attributed to atomic explosions.

———. Research on atmospheric radioactivity. Comptes rendus, v. 238, 1954: 2074–2075 (in French).

In flight at 2,800 meters, traces of radioactive particles were gathered by means of a corona effect similar to that of Sella, and of long period, on April 24, 1954. At Puy-de-Dôme (1,465 meters) after a fall of rain and snow on May 3–4, 1954, samples of the fallen residue showed very feeble radioactivity of period 10 days or more. This radioactivity of the airborne particles was independent of their probable natural electrical charge and mobility and was indicative of an atomic cloud.

———. Researches in radioactivity at the top of Puy-de-Dôme. Comptes rendus, v. 233, December 3, 1951: 1447–1448 (in French).

Snow collected at Puy-de-Dôme which fell on November 19–20, 1951, showed β-activity about 43 percent above background with an apparent half-life of 10 days. Speculation on role of particles as condensation nuclei.

———. Studies on the radioactivity of the atmosphere. Comptes rendus, v. 233 Oct. 15, 1951: 860–862 (in French).

The existence in the atmosphere of a radioactive substance, labeled "A," of several hours half life has been confirmed in flights at 3,300 meters, and at a ground station at 1,460 meters. Concentrations of Rn, Tn, "A," and "A' " observed from March 15 to August 14, 1951, are tabulated. The concentration of substance "A," of 20- to 30-hour half life, is related to atomic explosions and precipitation.

Lacassagne, A. Medical consequences of atomic bomb explosions. Bruxelles-Médical, (Brussels) v. 35, September 11, 1955: 1821–1833 (in French).

Martin, Charles Noel. Accumulative effects on the global surface caused by thermonuclear explosions. Comptes rendus, v. 239, 1954: 1287–1289.

HNO_3 is formed which locally lowers the pH of rain water. This can affect plant metabolism. C^{14} production and its absorption by living things is discussed.

Nahmias, M. E. Detection at a distance of atomic bomb tests. Mem. Artillerie Franc. 28, 1954: 393–402.

Nahmias describes the radioassay of atmospheric air, rain, and snowfall as the basis for detecting atomic bomb tests. Tables and graphs give time versus Ra equivalents, distance versus Roentgens/hours of radiation, and distribution of radio-elements which can be expected.

Ravina, A. The first known effects of hydrogen bomb on man. Presse médicale (Paris), v. 62, June 5, 1954: 881 (in French).

Tanaevsky, Olga and Vassy, Etienne. Variations of the natural and artificial radioactivity of the atmosphere. Comptes rendus, v. 241, 1955: 38–40.

The natural activity of the atmosphere, presumably Rn, Tn and their decay products, was most evident during periods of weak-winds at the Val Joyeux Scientific Station. In 30 of the 66 cases of high activity, the winds were from the southwest or west southwest, indicating a Rn source in that direction. The artificial radioactivity, detected in rains and snows, was strongest at the beginning of the precipitation. The highest activity measured was 0.724 microcuries/l.

GERMAN AND AUSTRIAN

Gerlach, Walther. The hazards of radiation and its danger to life. Universitas (Stuttgart, Germany), v. 2, 1957: 125–131.

Haxel, O., and Schumann, G. On the radioactive contamination of the atmosphere. Naturwissenshaften (Berlin), v. 40, 1953: 458 (in German).

Beginning on March 19, 1953, radioactivity in the air near Heidelberg was measured continuously by means of air filtering, using, in general, 48-hour exposures. The long-lived activity, presumably from atomic explosions, showed several peaks in the period from mid-March to mid-June, reaching a maximum of 2.5 curies/m^3 in mid-April. An examination of the decay rates allows the determination of the time of explosion. It was found that fission products reached Heidelberg in as little as 7 days from the Nevada test site.

Herbst, W., and others. Considerations of the suitability of radioactive atomic aerosols as tracers in meteorological flow investigations. Naturwissenschaften (Berlin) v. 41, 1954: 156–160 (in German).

A large increase in radiation from the ground at Wittental (Brunswick), October 18–20, 1951, led to an attempt to trace the increased radioactivity to atom bomb explosions in the United States and the corresponding measurements by Holter and Glasscock at Helena, Mont. For the explosions on October 6, 7, and 14 the probable track of air at the 500 mb level from Helena to Wittental was determined in each case and shown on a chart.

——— and Philipp, K. The path of an atomic explosion aerosol. Naturwissenschaften (Berlin), v. 40, 1953: 54 (in German).

Experiments at Wittental show that during October 16–24, 1951, a high value of the radioactive background was discovered. A similar high value had been reported at Helena, Mont., between October 6–16, 1951. It is suggested that the same air mass was at these two places at the different times and that radioactivity measurements permit the path of the air mass in which the explosion occurred to be plotted.

Sittkus, A. Observations on radioactive vapour from atomic experiments in the years 1953/54. Naturwissenschaften, (Berlin) v. 42, 1955: 478–482 (in German).

Records data from rainfall at Freiburg, Germany, on the radioactivity of the atmosphere and describes a method of deducing the time of the explosion from the observations.

———. The path of an atomic explosion aerosol. Remarks of Mr. Sittkus to W. Herbst. Naturwissenschaften, (Berlin) v. 40, 1953: 198.

No comparable effect was detected on other geiger counters in the same neighbourhood at the time when Herbst and Philipp recorded the effect they believe due to an atomic bomb. In reply these authors pointed out that they used thin-walled counters to detect β-radiation, whereas Sittkus used tubes suitable for detecting the penetrating component of cosmic rays.

Steinhauser, F. Atomic bomb explosions and weather events. Universitas (Stuttgart) 1954: 1189–1196 (in German).

———. Atomic energy and world weather. Universum natur and technick (Vienna) v. 16, 1954: 481.

———. Atomic energy experiments and weather. Osterreichische Hochschulzeitung (Vienna) 6 jg., Wien 1954 (in German).

Tsuzuki, M. Radioactive damage of Japanese fishermen caused by Bikini ashes. Münchener medizinische wochenschrift (Munich), v. 97, August 5, 1955: 988–994 (in German).

Short description of clinical experiences with radioactive injuries of the 23 fishermen during 1 year. All of the 23 fishermen in the boat were afflicted with acute radioactive-sickness as a result of contact with radioactive rain and ashes. They were injured through the combination of external as well as internal radiation.

INDIAN

Bandopadhyay, K. G., and others. Radioactive nuclei in rains over Calcutta. Science and culture (India), v. 21, 1955: 273–275.

The radioactivity in dusts carried down by rains between March and September 1955 were determined by β-ray assay. Histograms of relative activities as a function of specific rainfalls are given.

Chatterjee, Santimay, and others. Dating a nuclear explosion. Science and culture, v. 20, 1955: 403–404.

A method is proposed for dating nuclear explosions from the composite beta-decay curves of the radioactive dusts.

——— ———. Measurements on radioactive dusts over Calcutta. Science and culture, v. 20, 1955: 399–401.

A brief report of the measurements of the radioactivity of rain water samples collected in Calcutta from April 29 to the middle of July 1954, is presented. Measurements of energy and half lives indicated that the dusts originated from nuclear explosions.

——— ———. Presence of radioactive dusts over Calcutta. Science and culture, v. 19, May 1954: 570–571.

Beginning April 4, 1954, settled dust in Calcutta was analyzed for radioactivity; none was found until the first rain occurred on April 29.

ITALIAN

Neuwirth, R. Meteorological utilization of measurements of the artificial radioactivity of the air and precipitation. Geofisica pura e aplicata (Milan, Italy) v. 32, 1955: 147–158. (In German.)

German, French, and American measurements of the rainfall and air activity are being evaluated. For that purpose, trajectories from the experimental grounds for bomb tests in Nevada to Western Germany are drawn. By means of intermediate values, the test possibilities of air paths—first only scheduled—are given. The so-called deposit spaces and meridional circulations, which are significant particularly in divergence regions, prove to be of especial importance. The mechanism of activation of precipitation is discussed. A connection between the activity of precipitation and air masses could only be found in individual cases. But it seems that semitropical air air masses dispose of a higher specific activity in comparison with the polar air masses.

Radioactive precipitations caused by the experimental explosion of atomic weapons. Minerva medica (Turin) v. 46, March 31, 1955: 640–642 (in Italian).

Santomauro, L. and Cigna, A. First measurements of the radioactivity in atmospheric precipitations. Annali di geofisca (Rome), v. 6, 1953: 381–387.

Measurements conducted between February 1951 and November 1952 showed that nuclear-weapon tests at Las Vegas, Eniwetok, and Montebello were followed, 1, 2, and 3 weeks later, respectively, by an increase in the radioactive content of rain and snow falling in Italy.

Spena, A. The genetic problem in its relation to the use of atomic energy. Annali di medicina navale e tropicale (Rome), v. 61, July–August 1956: 569–582.

JAPANESE

Arakawa, Akio, and others. Climatic abnormalities as related to the explosions of volcano and hydrogen-bomb. Geophysical magazine (Tokyo) v. 26, 1955: 231–255.

The effects of volcanic explosion on climatic abnormalities are investigated statistically and synoptically. The abnormal weather during the summer season of 1954 is found to have features similar to climatic abnormalities caused by volcanic dust. The distribution of temperature anomalies and its annual variation are discussed in relation to the tropospheric circulation.

Arakawa, H. Abnormal weather caused by the H-bomb. Astronomy and meteorology, (Tokyo) v. 20, 1954.

———. Possible atmospheric disturbances and damages to the rice-crops in northern Japan that may be caused by experimentation with nuclear weapons. Geophysical magazine (Tokyo) v. 26, 1955: 125–134.

———, and Tsutsumi, K. A decrease in the normal incidence radiation values for 1953 and 1954 and its possible cause. Geophysical magazine (Tokyo) v. 27, 1956: 205–208.

Arizumi. On the distribution of ash-fall (meteorological investigation on the H-bomb experiment at Bikini Island—II) Journal of the Meteorological Society of Japan (Tokyo), Nos. 9–10, 1954.

Egawa, Tomoji, and others. Investigations on the contamination of field crops by artificial radioactivities as a result of the H-bomb tests at Bikini Atoll. Soil and plant food, v. 1, 1955: 19–20.

Crop samples taken between June and October 1954 were analyzed for radioactivity. Rare earth elements contributed the greater part of the activity. Polished rice showed no activity.

Horie, Kuniko. Damping of radioactivity of the Bikini ashes. Kagaku (Science) (Tokyo), v. 25, 1955: 636–637.

The radioactivity (β- and γ-radiation) of the H-bomb ashes was measured over a period of 600 days by means of an electroscope and a Geiger-Muller counter. Absorption by Al foils shows that the half-life is shorter for radiation of lower energy.

Ito, Gakuro and Moriuchi, Yasuyuki. Some problems on the radiological protection: especially concerning with the maximum permissible dose. Oyô Butsuri (Tokyo?), v. 24, January 1955: 3–17 (in Japanese).

Kakehi, H. Ash of Bikini and its effect on human body. Journal of Japan Physicians' Society, v. 31, May 1, 1954 (in Japanese).

Discusses physical and chemical composition of radioactive ashes which fell on the fishermen of the Fukuryu Maru and gives a clinical study of its effects. Estimated radiation received by fishermen in 2-week stay on ship as 200 r. Discusses hazard from contaminated tuna.

Kaneshige, Kankuro. Japan-United States radiobiological conference. Contemporary Japan (Tokyo), v. 23, Nos. 4–6, 1955: 296–310.

Kawabata, Toshihuri. Studies on the radiological contamination of fish. Japanese journal of medical science and biology (Tokyo), v. 8, October 1955: 337–372.

The decay rate of the radioactivity of the organs of fish caught by the crew of the "Shunkotsu Maru" was measured. After 3 months, the spleens, kidneys, and gonads retained considerable radioactivity, suggesting the presence of long-lived radioactive elements. At the end of this time, the liver and other organs retained only about 10 percent of the initial radioactivity and the bile had lost most of its activity. The retention by the pyloric caeca and the intestinal contents varied considerably and was probably dependent on the food consumed. Absorption of the ash of the organs on Dowex 50 and elution located most of the activity in the fractions eluted with 0.5 percent oxalic acid and 5 percent citrate buffers of pH 4.1 and 4.6. Qualitative separation with carriers identified the main radioactive element of the citrate buffer, pH 4.1, eluate as Zn^{65}.

Kimura, Kenjiro. Radioactive ashes on the fifth Fukuryu-Maru, the fishing boat that suffered from the hydrogen bomb test on March 1, 1954. Kagaku, (Tokyo) v. 24, 1954: 300–302.

By ordinary procedures with carriers and by separation with cation-exchange resins, the ashes were analyzed and the following radioactive nuclides were detected, Zr^{95} (65 days), Nb^{95} (35 days), I^{132} (2.4 hours), Te^{132} (77.7 hours), Nb^{95m} (90 hours), I^{131} (8.141 days), Ba^{140} (12.8 days), La^{140} (40.0 hours), Sr^{89} (53 days), Sb^{127} (93 hours), Ru^{103} (39.8 days), and Ru^{106} (1.0 year) etc.

———, and others. Detection of rhodium–103m in the "Bikini ashes." Bulletin of the Chemical Society of Japan, v. 29, 1956 (in English) 395–398.

The radiochemical analysis of the so-called Bikini ashes which fell on a Japanese fishing boat, the No. 5 Fukuryu Maru on March 1, 1954, are described as of some 25 days after detonation of the bomb. The collected sample (10^{-7} counts/minimum) was ignited and dissolved in 6N HCl, insolubles were filtered off, and the activity of small aliquots of the filtrate was measured. Total activity was estimated about 10^{-6} counts/minimum. Ru (10 mg.) was added to the filtrate as a carrier, the acidity of solution was adjusted to 2N, H_2S was passed through to precipitate R_u as sulfide, and the precipitate was dissolved with HNO_3; H_20, KM_nO_4, and concentrated H_2O_2. The appropriate aliquot portion of the distillate was taken up in a counting dish and evaporated to dryness; the activity was measured and found to be 1.5×10^{-5} counts/minimum.

Kosaka, Takao, and others. Radioactive rain and contaminated atmosphere observed in Niigata City (Japan). First report: the effect on environment and human body. Niigata Medical Association journal, v. 69, 1955: 1–6.

Koyama, Y. and others. Clinical course of the radiation sickness caused by Bikini ashes: intermediate Report. Iryo (Tokyo) v. 9, January 1955: 5–45 (in Japanese).

Clinical observations are summarized covering a 5-month period on 16 patients exposed to fallout from the Bikini explosion on March 1, 1954.

——— ———. Conference of the radioactive disease caused by the atomic bomb explosion in the central Pacific. Iryo (Tokyo) v. 9, January 1955: 56–68 (in Japanese).

Mitsui, Shingo, and others. Investigations on the radioactive contamination of crop plants as a result of hydrogen-bomb detonation. Soil and plant food, v. 1, 1956: 15–18.

I. Radioactive contamination of crop plants and soil. II. Root and foliage uptake of Bikini ash.

Miyake, Y. Artificial radioactivity in rain water observed in Japan, from autumn 1954 to spring 1955. Papers in meteorology and geophysics, Meteorological Institute, Tokyo, v. 6, May 1955: 26–32.

At about midnight of September 18, 1954, a typhoon (No. 14, 1954) ran away toward the sea after attacking Japan. In place of the typhoon a colder and less moist air flowed in from the north. Just at the time, a new activity of artificial origin was detected in rain water at Niigata and Hirosaki, both situated along the Japan sea coast of the northern part of the main island. From 22d to 24th of September, the activity increased rapidly, spreading over a wide area in Japan and finally an activity as strong as 0.3×10^6 curie/liter was counted in rain water at Yamagata.

———. The artificial radioactivity in rain water observed in Japan from May to August 1954. Papers in meteorology and geophysics, Meteorological Institute, Tokyo. v. 5, September 1954: 173–177 (in English).

Radioactivity in rainfall was measured at several places in Japan after the spring, 1954, Pacific tests. The maximum activity, 0.5×10^{-6} c/l, was observed at Kyoto on May 16, 1954. Meteorological trajectories indicate air that was over Bikini on May 8 reached Japan on the 16th and it is speculated that an explosion on May 5 is responsible for the activity. On August 3, airplane measurements with a dust impinger indicate $0.8 \sim 2.0$ x curie/cc on the average from 1000–3000 meters over Tokyo.

Miyake, Y. Rain from south and snow from north. Kayaku Asahi, (Tokyo) December 1954 (in Japanese).

Discusses detection of nuclear explosions by various methods including observations of fission product activity in the atmosphere. Deposition of 750 cpm on a vaseline coated paper (30×30 cm) on May 13–16, 1954. Eighty-six thousand cpm/l observed in rain at Kyoto on May 14, apparently from May 5 test at Bikini. Thereafter, strong contamination of rain observed at many places on Pacific Coast of Japan. Since May 1954, activity of rain on Pacific side about an order of magnitude greater than on Japan Sea side of Japan. On September 22, 1954, a record-breaking 124,000 cpm/l from a Russian test was observed in rain at Yamagata, associated with a cold front advancing from Siberia, almost no activity in warm front rain on the Pacific coast. Discusses possible hazard from contaminated snow.

——— and Sugiura, Y. Radiochemical analysis of radio-nuclides in sea water collected near Bikini Atoll. Papers in meteorology and geophysics, Meteorological Institute, Tokyo, v. 6, 1955: 33–37.

A radiochemical analysis of sea water containing fission materials collected near Bikini Atoll in June 1954, was performed. The sea water was boiled with hydrochloric acid; iron and lanthanum salts each 5 mg as Fe and La were added to it. They were precipitated as hydroxide, which was dissolved in hydrochloric acid and ferric chloride was extracted with ethyl ether. The remaining solution was evaporated to dryness and the residue was dissolved in hydrochloric acid. Using the latter solution the group separation was done with cation exchanger resins.

———, and others. Artificial radioactivity in the sea near Japan. Papers in meteorology and geophysics, Meteorological Institute, Tokyo, v. 6, May 1955: 90–92.

Sea water collected around the Bikini Atoll from July to September 1954, was analyzed for total radioactivity by adding 2 g. solid NH_4Cl, 1 ml. of an aqueous solution of Ferric alum (86.3 g./l.), and 1 ml. of $BaCl_2$ solution (17.8 g./l.) to 1 l. of H_2O heated to 60–70 while being stirred. NH_4OH was added until the solution was faintly pink to phenolphthalein. After 2-minutes boiling the precipitate settled on standing for several hours at room temperature before being filtered on a filter disk lain above a glass filter. Counting rates of 2.1 ± 1.6 to 140.8 ± 6.8 counts/minute/l. were obtained.

——— ———. On the distribution of radioactivity in the sea around Bikini Atoll in June 1954. Papers in meteorology and geophysics, Meteorological Institute, Tokyo, v. 5, January 1955: 253–263.

Report of an oceanographic survey in the late spring of 1954 in the Marshall Islands area to investigate the radioactivity of the waters following the Castle tests. Maximum value was 7025 cpm/l, 450 km west of Bikini at a depth of 75 m (1,000 cpm ≈ 5.9 mμc). Almost all radioactivity was in solution, a filter of pore size 0.5μ passed 99 percent of the activity. Distribution of the radioactivity and its relation to ocean currents is shown. Vertical cross sections show marked decrease in activity below thermocline, about 150 m. Estimate flow of radioactivity through cross-section 150 km west of Bikini was 10^8 curie/hour. Coefficient in mixed fission product decay law ranged from −1.8 to −1.6, mean −1.5.

——— ———. Radiochemical analysis of fission products contained in the soil collected at Tokyo, May 1954. Papers in meteorology and geophysics, Meteorological Institute, Tokyo, v. 6, 1955: 93–94.

Soil (300 g.) was leached with 50 ml. 6N HCl on a steam bath and the filtered solution evaporated to dryness. The residue was dissolved in distilled water and an aliquot of the solution was subjected to chemical analyses, in which the sample was dried on a stainless steel planchet and its β-rays were counted. Group separation of the extract was made after addition of carriers of Ce, Ba, and Sr. Precipitation with H_2S showed very weak activity which was only a few percent of the total. The hydroxide group contained an appreciable amount of radionuclides, but most of them were insoluble when changed into fluoride forms. The filtrate of fluoride solution also showed a weak activity. The radionuclides obtained in the carbonate fraction were separated into Ca, Sr, and Ba and the Ca fraction was separated by concentrated HNO_3 and the Ba fraction obtained by precipitation as chromate. Results show radionuclides of rare earths $= 9 \times 10^{-13}$ curie/g. $Sr^{89} = 3 \times 10^{-13}$ curie/g., and $Ba^{140} = 7 \times 10^{-13}$ curie/g.

Ohashi, S., and others. Pathological findings in the fatal case (the late Mr. Kuboyama) of the radiation sickness caused by Bikini ashes. An intermediate report. Iryo (Tokyo), v. 9, January 1955: 46–55 (in Japanese).

Autopsy findings and the case history are summarized from a case diagnosed as radiation sickness caused by exposure to fallout from a thermonuclear explosion. The patient died 207 days following exposure while on a fishing boat said to be located about 100 miles east of Bikini at the time of the explosion. Evidence was also found of a secondary virus hepatitis and aspergillus fumigatus pneumonia.

Otsuka, R. and Shimada, K. On the upper air current in lower latitude of the north western Pacific ocean at the beginning of March 1954. Meteorological investigation on the H-bomb experiments at Bikini Island. Journal of the Meteorological Society of Japan, v. 32, No. 7–8, 1954.

Ota, Michio, and others. Contamination of grapes by radioactive substances. Soil and plant food (Tokyo) v. 1, 1955: 43–44.

The K content of grapes was determined by measuring K^{40} content from 1951 to 1954. After the radioactive fallout in 1954, the grapes were shown to be contaminated by radioactivity.

Obo, Fujio. Radioactive rains and fishes in the Kagoshima area. Medicine and biology (Tokyo) v. 33, 1954: 19–23 (in Japanese).

Results are given of radioactivity determinations of rains, well and city water, vegetables, domestic animals, milk, and fishes. Radioactivity was determined in a radiation counter (Scientific Research Lab. model 32), at a distance of 1 cm. for 10 minutes. Samples were obtained during 18–27 May, 1954. The highest and lowest values obtained were: 4000–80 counts/min/1/ (c. p. m.) for rains, 20–0 c. p. m./cc. for well water, and 71–0 c. p. m./100 cc. for city water.

Radioactivity in the pelagic fish. Bulletin of the Japanese Society of Scientific Fisheries (Tokyo) v. 20, 1955: 907–926.

I. Distribution of radioactivity in various tissues of fish. Bulletin of the Japanese Society of Scientific Fisheries, v. 20, 1955: 907–915.

Pelagic fishes caught after atomic explosion experiment at Bikini Atoll in the Pacific were examined by radiochemical techniques. Generally the radioactivity was large in liver, kidney, gall bladder, and heart, and then in pyloric caeca, stomach, intestine, and gonad: there was little activity in skin, bone, and muscle. This order varied with species. Large radioactivity of the stomach contents did not necessarily mean large activity in the tissues, indicating considerable participation of diffusion of sea water into the fish body. Muscles from various sites showed slight difference in the activity. The dark muscle, however, showed several times as large activity as ordinary muscle.

II. Group separation of radioactive elements in fish tissues: p. 916–920.

Analytical group separation was performed with various ashed tissues of some fishes exposed to radioactive ash. The radioactivity was particularly large with element belonging to the third group, both A and B subgroups The second group showed considerable activity in pyloric caeca and kidney of skipjacks. The radioactivity of the first and fourth groups was detected in some tissues; the fifth group showed slight activity.

III. Separation and identification of zinc 65 in the muscle of skipjack.

Muscles of skipjack caught in the vicinity of the Bikini Atoll after the explosion were ashed, treated with Dowex 50, and eluted with various solvents. A fraction obtained with 0.5 percent oxalic acid and ammonium citrate (pH 4.18) contained Zn^{65}.

Saiki, Masamichi, and others. The radioactive material in the radiologically contaminated fishes caught in the Pacific Ocean in 1954. Bulletin of the Japanese Society of Scientific Fisheries (Tokyo) v. 20, 1955: 902–906.

The radioactivity of several samples of Coryphaena Hippurus caught in the southern Pacific in May 1954, after the atomic explosion at Bikini, was found, in decreasing order, in spleen, kidney, liver, pyloric caeca, heart, gill, intestine, gastric wall, ovary, testis, gastric content, red muscle, skin, vertebrae, and muscle. The red muscle of Neothunnus Macropterus showed 54.8 counts/min./0.20 g. activity on dry basis; the activity was decreased to 27.6 by soaking 25 g. muscle in 25 cc. water, and to 14.1 by soaking in 0.5 percent Na ethylenediaminetetraacetate solution. The radioactive substances in these fish tissues were found, upon analysis, to belong to the III group, particularly to III–B group. Examination of synchroscope patterns by scintillation counter indicated the presence of Zn^{65} among the radioactive substances. Sr^{90} was suggested to be present in very small amount.

Shimizu, Kentaro. Hiroshima and Bikini. Oriental economist, (Tokyo) v. 22, July 1954: 344–346.

A report by the doctor who treated the Japanese fisherman injured by the Bikini test.

Shinjiro, T. Death ash, experience of 23 Japanese fishermen. Japan quarterly, v. 2, 1955: 37.

Tajima, Eizo. Why fishing boats were contaminated by radiation. Shizen, December 1954 (in Japanese).

Many Japanese fishing boats were examined with a G–M counter following the Bikini tests of 1954. Decks and other washable parts were weakly irradiated, lamps and other unwashable parts were strongly irradiated. Directional relationships of contaminants on individual ships coincided with those of the prevailing winds. Ships to the west of Bikini averaged 123 cpm; those to the east, 1,800 cpm. Activity to the east shows sharp rises on test dates, sharp drops on other dates. To the west of Bikini, a strip of water from 15° N. to about the equator is contaminated, and the contamination of the boats may have been due to this.

Takase, Akira. Distribution of radioactivity in various tissues of fish and group separation of radioactive elements in them. Bulletin of the Institute of Public Health (Tokyo), v. 4, 1955: 27.

Radiological studies were made of several kinds of fish which had been caught in the fishing ground including the area from longitudes 128° East to 162° East and from latitudes 3° North to 33° North during the period from April 25 to July 7, 1954, and which had been rejected as highly contaminated radiologically at the time of landing.

———. Separation of the radioactive elements in the muscle of skipjack by ion-exchange resin, and confirmation of the presence of radioactive zinc. Bulletin of the Institute of Public Health (Tokyo), v. 4, 1955: 22–26.

An ashed sample of skipjack muscle caught in June 1954, near Bikini Atoll was analyzed for elements separated by an anion-exchange method (Dowex 50) with the use of 0.2N HCl, 0.5 percent oxalic acid, and 2 percent NH_4 citrate as eluents at each pH value of 3.53, 2.18, 4.60, 5.02, 5.64, and 6.42.

Yamada, Yoshio, and others. Measurement of radioactivity in contaminated crops. Soil and plant food (Tokyo), v. 1, 1956: 25–26.

A method called the direct method was developed to correct for natural K^{40} radiation in plant samples. The K_{20} content of the ashed sample is determined by flame-photometry. The radioactivity in a 100-mg. sample is measured and the natural radioactivity from K^{40} determined by calculation subtracted. Tea samples tested gave evidence of contamination by radioactive fallout.

Yamamoto, Ryozaburo. Atmospheric oscillation caused by the H-bomb. Astronomy and meteorology (Tokyo), v. 20 No. 8, 1954.

Yamasaki, F., and Koneko, H. On the artificial radioactivity in rainwater. Journal of the Scientific Research Institute (Tokyo), v. 49, June 1955: 137–143.

Rainwater in Tokyo was examined for artificial radioactivity from April to December 1954. The most active rain occurred on May 17, as reported by Miyake. Wide variability in activity was noted from sample to sample, even in the same rainfall. In a single rainfall, the specific activity appeared to be negatively correlated with rainfall intensity. Rainwater collected in the early stage of a single rainfall did not always show the strongest radioactivity. Exponent in the decay law for mixed fission products ranged from -0.9 to -1.4 in five rain samples investigated.

Yatazawa, Michihiko, and Ishihara, Takashi. Radioactive contamination of plants in Japan covered with fallout from H-bomb detonations in March–May 1954 at Bikini Atoll, Marshall Islands. I. Distribution of deposited radioactivity. Soil and plant food (Tokyo), v. 1, 1955: 21–22.

In May 1954 rains contained radioactivity up to 0.2 muc. per liter. The provisional permissible level of unknown radioisotopes in H_2O is given as 10^{-7} muc./ml. for $\beta-$ or $\gamma-$ emitters. The safety factor for these values is at least 100. From these values the permissible level for foods was calculated as 0.22 muc./day. Food plants tested ranged 0–1.25 muc./10 g. dry matter. It is concluded that serious radioactive contamination of plants was probable.

RUSSIAN

Marei, A. N. Radioactive wastes and public health problems. Meditsinskaia radiologiia (Moscow) v. 1, July–August 1956: 3–7 (in Russian).

Yakobson, I. I. Initial radioactive investigations in Russia. Akademiya Nauk Uzbek S. S. S. R., v. 5, 1953: 118–135.

SWEDISH

Muller, Hermann J. The manner of dependence of the permissible dose of radiation on the amount of genetic damage. Acta radiologica (Stockholm), v. 41, January 1954: 5–20.

Morgan, K. Z. Maximum permissible concentration of radioisotopes in food, water and air and maximum permissible equilibrium amounts in the body. Acta radiologica v. 41, January 1954: 30–46.

Warners, C. J. Note on radioactive compounds in the atmosphere. Tellus (Stockholm) v. 7, August 1955: 403–404.

Since January 1, 1955, daily measurements of the radioactivity of the air have been made by the Royal Netherlands Meteorological Institute. The highest values noted were in the period April 28–30, in tropical air transported from the south, and reached 27.4×10^{-10} μc per liter.

OTHER NATIONS

Akpinar, S. and Akpinar, R. Radioactive precipitations in Istanbul and Uludag (Turkey) Istanbul, v. 20C, 1955: 287–302 (in English).

From the decay curves of fission products in rain and snow, it was possible to determine the date of announced United States atomic tests and unannounced Russian tests with a probable maximum uncertainty of ± 8 days.

Gabites, J. F. Drift of radioactive dust from the British nuclear bomb test in October 1953. New Zealand Journal of science and technology (Wellington, N. Z.) v. 36B, September 1954: 160–165.

To account for the second wave of radioactivity which reached Wellington 38–54 hours after the blast in Woomera, Australia, it is apparently necessary to assume the material started from a height of 22,000–25,000 feet. The dust was dispersed downward by eddy diffusion and carried by lower level atmospheric circulations to New Zealand. The first wave which occurred 30 hours after the blast remains unexplained.

Holubec, K. Lesson of Hiroshima and Bikini. Časopis lékařů českých (Prague) v. 95, May 11, 1956: 518–525 (in Czech).

Levi, H. Natural background radiation and radioactive fallout. Ugeskrift for laeger (Copenhagen) v. 117, December 15, 1955: 309–311 (in Danish).

Piédrola, Gil G. and Amaro, Lasheras J. Precipitation of radioactive dust (fall-out); necessity for a national detection and defense organization. Medicina colonial (Madrid) v. 28, October 1, 1956: 229–231 (in Spanish).

Rose, D. C. and Katzman, J. Radioactive deposits found at Ottawa after the atomic explosions of January and February 1951. Canadian journal of physics, v. 30, March 1952: 111–116.

Gamma-ray measurements indicated that considerable radioactive matter, apparently consisting of fission products, fell on January 29, and on February 7, 1951. The earlier fall came from an explosion in Nevada on January 27, and the February 7 material appeared to be 3 to 5 days old. The quantity was estimated to be equivalent to 1 γ Ra per square mile.

Ryder, N. V., and Watson-Munro, C. N. The detection of radioactive dust from the British nuclear bombs of October 1953. New Zealand journal of science and technology (Wellington, N. Z.), v. 36B, September 1954: 155–159.

Thirty hours after the nuclear explosion near Woomera, Australia, in October 1953, a peak in β-activity of 50 cpm was observed in Wellington, New Zealand, on an air filter. A second peak of 6 cpm was observed a few hours later. Decay curves indicated the activity was fission product activity.

Szalay, A. Unusual radioactivity observed in the atmospherical precipitation in Debrecen (Hungary) between April 22–December 31, 1952. Acta physica (Budapest, Hungary) v. 5, 1955: 1–14.

The authors investigated, by means of an end-window β-counter tube equipment the activity of precipitation fallen in Debrecen between April 22 and December 31, 1952. At times the precipitations showed radioactivity which proved to be due to fission products deriving from atomic explosions. These anomalous activities, with a lag of a few days, were in correlation to time with the atomic explosions published during the same period.

Popular Periodical Articles

A-bomb survivors due to lose quarter of life. Science news letter, v. 69, March 31, 1956: 201.

All about A-bombs, fallout, dangers in the future. U. S. news and world report, v. 38, April 29, 1955: 96ff.
Extracts from transcript of hearings held by the Joint Committee on Atomic Energy, April 15, 1955.

Amrine, Michael. Atomic clouds over America. Science digest, v. 33, June 1953: 23–30.

———. Fallout, can man survive? Progressive, v. 21, February 1957: 6–10.
The author is deeply concerned about the dangers of radioactive fallout from bomb tests, and questions the administration's apparent lack of concern.

Armagnac, A. P. How the H-bombs spread radioactivity. Popular science, v. 166, April 1955: 144–145.

———. Will bomb dust endanger your health? Popular science, v. 170, February 1957: 163–167.

Arnold, James R. Effects of the recent bomb tests on human beings. Bulletin of the atomic scientists, v. 10, November 1954: 347–348.
"The death of a Japanese fisherman on September 23 [1954] not only shocked the world but made the radioactive fallout seem the most fearful consequence of the H-bomb."

As the bomb goes off and the cloud heads east * * * science tackles radiation peril. Life, v. 38, March 21, 1955: 32–39.

Ascoli, Max. There must be an end to it (editorial). The Reporter, v. 16, May 16, 1957: 8–9.
Urges end of bomb testing.
Reprinted in extension of remarks of Charles O. Porter, Congressional record [daily edition] v. 103, May 13, 1957: A3637–A3638.

Atom outgrows its proving ground. U. S. news and world report, v. 36, March 26, 1954: 45–47.
"Thousands of square miles now can be damaged by radioactive particles, from one H-bomb, carried by the wind."

The atom: "Unpleasant debate." Newsweek, v. 48, November 26, 1956: 64–66.
Various experts in the atomic energy field comment on dangers of strontium 90.

Atomic radiation: the r's are coming. Time, v. 58, June 25, 1956: 64f.

Ban the "dirty bomb," (editorial). New Republic, v. 136, April 29, 1957: 3–4.
Proposal that the United States should seek an international agreement to limit the radioactive debris from bomb tests.

Bengelsdorf, Irving. Can the atom change the weather? Saturday review, v. 39, July 7, 1956: 31–37.

Berninger, Karl. The bomb and the weather. Contemporary issues (London) v. 6, March-April 1955: 114–116.
Maintains that atomic tests do influence weather conditions; points out meteorological phenomena to bolster this thesis.

Berrill, Norman J. A Canadian scientist asks have we gone too far with the atom tests? Maclean's magazine (Toronto) v. 68, July 9, 1955: 7–9 ff.
"Every time an atomic explosion occurs anywhere in the world more highly dangerous radioactive particles are set free. Professor Berrill says it's time our leaders told us the truth—that even if we survive we may breed a future race of morons or monsters."

———. The menace of radiation. Atlantic monthly, v. 196, October 1955: 49–54.
"Radioactive fallout from test explosions of atomic bombs has made clear to Americans that nuclear warfare would mean annihilation of large areas. Less well understood is the fact that leakage of radioactive materials resulting from careless operation of atomic powerplants and other peacetime uses of nuclear energy can be just as deadly."

Bishop, R. Can fish survive the atom? Field and stream, v. 61, June 1956: 70–71.

Blifford, Irving H. Total radioactive fallout. Science news letter, v. 69, April 28, 1956: 267.

Bomb watchers; radioactive dust in Japan. Time, v. 67, April 16, 1956: 56 f.

Bombs and the species. Economist, (London) v. 175, May 14, 1955: 557–558.

Cattle caught in fallout cancerless. Science news letter, v. 71, April 20, 1957: 248.

Danger, strontium 90. Newsweek, v. 48, Nov. 12, 1956: 88 f.

Data on atomic radiation transmitted to U. N. committee. United States Department of State bulletin, v. 35, October 29, 1956: 687.

Davis, Helen M. Hazards of smog. Science news letter, v. 67, May 7, 1955: 298–299f.

"The problem of preventing harmful radioactive fallout is like that of smoke control. Filters and precipitators can reclaim valuable wastes from industrial chimneys."

De Roos, Robert. What are we doing about our deadly atomic garbage? Collier's, v. 134, August 20, 1954: 28–34.

Espinasse, Paul G. Biology and the bomb. Nation, v. 180, June 25, 1955: 579–581.

Despite the comforting statements of AEC on the background radiation increase caused by atomic tests, this author points out that a very little upset in the "balance of nature" might have very serious ramifications.

Facts about A-bomb fall-out. U. S. news and world report, v. 38, March 25, 1955: 21–26.

"Not a word of truth in scare stories over tests."

Abridged in Reader's digest, v. 66, June 1955: 22–24.

Fallout detector developed by Navy, Army-Navy-Air Force register, v. 76, December 10, 1955: 6.

Fallout hazard to grow; strontium–90. Science news letter, v. 71, February 23, 1957: 115.

Ferreus. Courage or perdition? The 14 fundamental facts of the nuclear age. Review of politics, v. 16, October 1954: 395–411.

Each fact is discussed in the light of its relationship to future policy.

Foulks, J. G. Do H-bomb tests threaten human survival? Canadian chemical processing, v. 39, April 1955: 29–30 f.

Glass, Bentley. The hazards of atomic radiations to man, British and American reports. Bulletin of the atomic scientists, v. 12, October 1956: 312–317.

Discussion of reports issued by National Academy of Sciences and British Medical Council on effects of radiation.

H-bomb contamination. Science news letter, v. 67, February 26, 1955: 134.

H-bomb such as that exploded in Marshall Islands would contaminate 7,000 square miles with radioactivity.

Turbulence in the atmosphere could make the area threatened twice as wide as the cigar shape (up to 40 miles) described in official AEC report.

H-bombs without fallout (editorial). Bulletin of the atomic scientists, v. 12, September 1956: 234ff.

Claims that statements on minimum fallout from H-bombs confuses public thinking.

Hayasaka, I. Appeal to the western evolutionists against the hydrogen bomb by the Japanese Society for the Study of Organic Evolution. Science, v. 120, October 8, 1954: 9A.

Same: Bulletin of the atomic scientists, v. 11, January 1955: 34.

Hicks, C. B. How dangerous is fallout? Popular mechanics, v. 106, November 1956: 198–202ff.

Holmes, Robert H. Latest about after-effects of A-bomb. U. S. news and world report, v. 38, May 13, 1955: 60–68.

The director of the Atomic Bomb Casualty Commission describes the present situation of the Hiroshima survivors, the effects of the bombing upon their offspring, and the present condition of Hiroshima itself.

Holzman, B. Have atom bomb tests fouled up the weather. Look, v. 17, August 11, 1953: 32–33.

Hot stuff: big hurdle for atom power. Business week, July 23, 1955: 72–82.

Problem of radioactive wastes discussed.

How dangerous are the bomb tests? Time, v. 69, June 3, 1957: 62, f.

How radiation can affect our children. Challenge, v. 5, October 1956: 54–59.

Interview with Warren Weaver, chairman, Committee on Genetic Effects of Atomic Radiation, National Academy of Sciences.

How safe humans? Newsweek, v. 49, February 18, 1957: 71.

Humanitarian bombs; minimum widespread fallout. New republic, v. 135, July 30, 1956: 3–4.

Inglis, David R. Prospects for stopping nuclear tests. Bulletin of the atomic scientists, v. 13, January 1957: 19–23.

A fervent plea for the ending of thermonuclear tests. The author believes the chief reason for ending them is not the radioactive fallout problem as much as it is the vital need to get on with some sort of disarmament.

Jacobs, Paul. Clouds from Nevada; a special report on the AEC's weapons-testing program. The Reporter, v. 16, May 16, 1957: 10–29.
Lengthy article describing the hazards to people living near the atomic testing grounds in Nevada.

Katz, Sidney. How serious is the threat of radiation? Maclean's magazine (Toronto), v. 69, December 8, 1956: 18–19, 115–118.

Knapp, H. A. South Woodley looks at the H-bomb. Bulletin of the atomic scientists, v. 10, October 1954: 306–311.

Lang, Daniel. Fallout. New Yorker, v. 31, July 16, 1955: 31–41.

Lapp, Ralph E. Civil defense faces new peril. Bulletin of the atomic scientists, v. 10, November 1954: 349–351.
Assessment of fallout and its impact upon civil defense. Urges that the Federal Government release classified data on fallout to provide guidance to civil defense organizations.

———. Confused alert. Life, v. 38, June 27, 1955: 48–49.

———. Fallout and candor. Bulletin of the atomic scientists, v. 11, May 1955: 170f.

———. Global fallout. Bulletin of the atomic scientists. v. 11, November 1955: 339–343.
Following a review of the fallout literature, the author concludes that, although many in the Pentagon maintain that blast and heat are the real military effects of nuclear weapons, it appears that local fallout from the uranium superbomb is a potent weapon. Remote fallout from even a large scale nuclear bombing, however, does not appear to be a global hazard to humanity.

———. Humanitarian H-bomb. Bulletin of the atomic scientists, v. 12, September 1956: 261–264.
"The superbomb can be designed to be relatively clean [by giving most of the energy to fusion, which produces nonradioactive helium] or very dirty [by maximizing fission]. The former would be desirable in the test series whereas the latter would seem to fulfill the requirements of a strategic weapon.

———. Radioactive fallout. Bulletin of the atomic scientists, v. 11, February 1955: 45–51; June 1955: 206–209.

———. Radioactive fallout. New Republic, v. 132, February 14, 1955: 8–12.
Discussion. *Ibid.*, v. 132, April 4, 1955: 23.

———. Strontium limits in peace and war. Bulletin of the atomic scientists, v. 12, October 1956: 287–289, ff.
Questions whether MPC figures released by AEC are in fact safe.
Reply: New republic, v. 135, October 15, 1956: 5–6.

Laurence, William L. Promise of tomorrow. Colliers, v. 136, December 23, 1955: 46, ff.

Libby, Willard F. The radioactive fallout. Civil defender, v. 1, December 1955: 16.
Speech given at Annual Meeting of National Association of State Civil Defense Directors, November 3, 1955.

———. Radioactive fallout. National safety news, November 1955: 60, ff.
Remarks prepared for delivery at the fourth annual conference of the United States Civil Defense Council, Hotel Statler, Boston, Mass., September 29, 1955.

———. Radioactive fallout, with editorial comment. Bulletin of the atomic scientists, v. 11, September 1955: 256–260.
Speech given before Alumni Reunion at the University of Chicago, June 3, 1955.

———. Radioactive fallout and radioactive strontium. Science, v. 123, April 20, 1956: 657–660.
Based on a speech given at Northwestern University, Evanston, Ill., January 19, 1956.
The radioactivity produced in magaton weapons is placed largely and immediately in the stratosphere while the smaller kiloton weapons produce clouds which do not reach into the stratosphere, and the bulk of their radioactivity is left in the troposphere. In the troposphere where rain occurs, particulate matter will be washed down in an period of days or weeks. It is easy to show that one-tenth inch of ordinary rainfall will probably remove almost completely all particulate matter except that which is so small as to be nearly of molecular dimensions.
Also in Ordnance, v. 41, July–August, 1956: 40–43, with title: Fallout from the A-bomb.
Reprinted in Congressional Record [daily edition] February 20, 1956: A1577–A1579.

Libby, Willard F. What the atom can do to you and for you. U. S. news and world report, v. 42, May 17, 1957: 64–75.
A member of the Atomic Energy Commission gives "authoritative" answers to such questions as: Should we worry over fallout from atomic tests? When will we get usable atomic power? Are new atomic weapons in sight?

———, and others. Forum on the Schweitzer declaration. Saturday review, v. 40, May 25, 1957: 8–13, 35–36.
There are four articles in this forum concerning atomic tests and the dangers of the resulting radiation. One is Dr. Libby's answer to the protest of Dr. Albert Schweitzer.

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Two scientists back tests of H-bombs, New York Times, November 6, 1956: 45.

Dr. Edward Teller and Dr. Ernest Lawrence declared "The radioactivity produced by the testing program is insignificant."

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Three proposals laid before U. N. General Assembly's 80-nation Political Committee a few hours after AEC reported that the Russians had set off at least their fifth nuclear explosion since August.

1. Japan, Canada and Norway: Called for advance registration of all nuclear tests and for observation of the radiation results by U. N. experts.

2. Sweden: Proposed that all nuclear explosions be banned until the U. N. Scientific Committee on the Effects of Radiation had completed studies now in progress. This would amount to a 2-year moratorium.

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Routine and special observations were made at Brookhaven on the radioactivity in settled dust and rainwater during the Nevada tests in the spring of 1953, together with some meteorological interpretation. It was estimated that 30.2 curies of activity were deposited on the 25.9×10^6 square mile Brookhaven site during the 4-month period. Results of various monitoring devices are given. Rain samples were investigated for solubility of fission products. Using Whatman–41 filter (efficient down to 0.7μ) 27.5 percent of

activity in residue, 72.5 percent in filtrate. Using Whatman–41 and HA molecular filter (efficient down to 0.2μ) 83.4 percent of activity in residue, 16.6 percent in filtrate, indicating significant portion of activity on particles between 0.2 and 0.7 μ. Other studies on solubility of fallout and on variation in rainout during course of storm, also gamma energy spectrum of rainout.

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An increase in radioactivity was noted in routine Brookhaven surveys in the second week of November 1952. Radioautographs showed little evidence of specific centers of activity but showed slight clouding of the film after 4 days' exposure, indicating that most of the particulate activity was from very small particles. Total activity estimated to have fallen on the 25.9×10^{10} cm^2 laboratory site during the 2-month period, November–December 1952, was:

	Curies
Settled dust, solid residue	.04
Settled dust, filtrate	.14
Precipitation, solid residue	.22
Precipitation, filtrate	.05
Total	.97

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A description of tumors on *Ipomoea tuba* from the A-bomb test sites on Eniwetok Atoll; Appendix to radiobiological survey of Bikini, Eniwetok, and Likiep Atolls, July–August 1949, by Susann F. Biddulph and Orlin Biddulph. Oak Ridge, Tenn., United States Atomic Energy Commission, Technical Information Service, [1952] 24 pp. (AECD–3446 App.).

Tumors on plants of *Ipomoea tuba*, a vine with large heart-shaped leaves, found in an area 400 to 600 yards from the bomb crater on an Eniwetok atoll, 17 months after an atomic explosion, are described in detail. After careful consideration of all possible casual agents, it was concluded that the tumors were radioinduced.

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In 3 out of 23 flights at 6,000 meters over France in July 1946, an unknown radioactive material with a half-life of 25 ± 5 hours was detected. Average concentration 0.013×10^{-16} curies/cm^3 radio equivalent. The first detection was on July 20, 1946, 20 days after the first explosion at Bikini. Similar material was detected in 6 out of 15 flights in July, 1948, the first on July 2, 75 days after the Eniwetok test.

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Incident of the Bikini ashes and the fishing boat is reported. Experiences on the boat are recorded, and fallout analyses are compared with those of Nagasaki and Hiroshima.

———. Radiochemical analysis of "Bikini ashes" fallen on board the No. 5 Fukurya Maru on March 1, 1954: pp. 7–27.

Comprehensive analysis was done in order to find the proper method of medical treatment for the victim fishermen on board. Analysis was started on March 18, and ash was found which consisted mostly of $Ca(OH)^2$, activity of which was 0.37 mc./g. on April 23. Cations of the third group (especially rare-earth metals) and fifth group were found to have strong activity by chemical separation. Fractions of each group, anions, Zr and Nb fraction, and U fraction were separated by an ion-exchange method.

Shiokawa, Takanobu, and others. Radiochemical studies on "Bikini ashes" (March 1, 1954): pp. 28–42.

Decay characteristics of the ashes which were brought back by the crew of the Fukuryu Maru No. 5 were; untreated ash $I = ct^{-1.81}$, water solution part $t^{-2.71}$ insol. part $t^{-1.68}$. Radioactive species separated by chemical method with carrier or collector were; nuclide, activity of nuclide (counts/min.) /activity of original sample (counts-min.), and the date of separation, Sr^{90} $6{,}000/80 \times 10^4$, April 24; Zr^{95}, $280/80 \times 10^4$, –; Ag^{111}, $200/200 \times 10^4$, April 14; Ru^{103}, $2.300/25 \times 10^4$ etc.

Yamatera, Hideo, and others. Radioactive dust from No. 5 Fukuryu Maru: pp. 43–54.

Analyses of radioactive dust collected on board No. 5 Fukuryu Maru were done by chemical separation and measurement of γ-ray energy and half-life of each species. Results are summarized as follows, radioactive nuclide and approximate percent of radioactivity given: Ru^{103}, 4.3–57; Ru^{106}, 1.4; Te^{129}, 1.3; I^{131}, 4.5; I^{132}, 1.0; Te^{132}, 1.0; etc.

Kiba, Toshiyasu and others. Radioactive substances found on the contaminated fish: pp. 55–60.

Radiochemical investigation was done on the substance collected from the surface of tuna fish which were brought back by the No. 5 Fukuryu Maru. Most of radioactivity was found on the scales, which could not be decontaminated by treating with H_2O; 80 percent of activity was removed by washing dried scales with 3N HCl. Paper chromatographic separation of the HCl fraction showed the presence of Ba^{140}, Sr^{89}, Te^{132}, and probably Zr^{95}, La^{140}, and rare earths.

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Shimizu, S., and others. Radioactive dust from nuclear detonation. Survey of the radioactive contamination of the No. 5 Fukuryu Maru: pp. 1–3.

A collection of reports on investigations on No. 5 Fukuryu Maru, a fishing ship which was in the vicinity of the Bikini atoll when nuclear detonation occurred on March 1, 1954. The radiation dosage rate of contamination observed for combined β- and γ-radiation at every part of the ship on March 19, April 21, and May 16 is recorded. The av. value of total γ-dosage for the crew was supposed to lie between 200 and 500 r.

Kikuchi, Takehiko, and others. Properties and size of the radioactive ashes obtained from the No. 5 Fukuryu Maru: pp. 4–11.

Size and radioactivity of the ashes collected from the ship have been measured. Ashes consist of particles which appeared dark when observed through an occular microscope. When observed by side illumination the particles appeared white and several black spots were seen on the surfaces.

———. Radioautographic studies of the radioactive ashes obtained from the No. 5 Fukuryu Maru: pp. 12–17.

Radioautographic studies have been made of the radioactive ashes obtained from the ship by the use of X-ray film, radioautographic stripping plates, and plates of α-emitters. The radioactivity was found not proportional to the size of the particle, and the distribution of radioactivity in each particle was not uniform.

———. Radioautographic studies of the materials obtained from the No. 5 Fukuryu Maru contaminated by radioactive ashes: pp. 29–34.

The contamination was associated with the presence of small radioactive particles. Although these particles were easily scattered, it was difficult to remove them completely. The particles did not penetrate into the interior of clothes of fine meshes. Decontamination by washing with sea water was not perfect.

———. The contamination of the fishes caught by the No. 5 Fukuryu Maru and the foods manufactured from these fishes: p. 35–38.

The radio-contaminated tunas and other fish caught by the ship in the vicnity of Bikini Atoll were studied. The contamination was caused directly by radioactive ashes and was limited to the surface of the fish. No radioactivity was detected in muscles and bones. The contamination of tuna expressed as Co^{60} was 10^{-2}—10^{-3} microcurie per square centimeter of skin and 10^{-1} microcurie per g. scales.

Ishibashi, Masayoshi, and others. Radiochemical analysis of the Bikini ashes: pp. 35–39.

The following nuclides were detected in the Bikini ashes by radiochemical procedures: Ca^{45}, Sr^{89}, Y^{91}, Zr^{95}, Ru^{103}, Nb^{95}, Rh^{103m}, Ru^{106}, Te^{129}, I^{131}, Ba^{140}, La^{140}, Ce^{144}, Pr^{144}, and U^{237}. The ion-exchange method was used for analysis of contaminated rain water which fell on the Kyoto area on May 16, 1954 from which the presence of Sr^{89}, Zr^{95}, and Ba^{140}, was detected. Rare earths seemed also to be present.

———. Analysis of carrier-free radioisotopes by paper chromatography: pp. 60–74.

Rf values of Ru-Rh, Zr, Nb, Y, Ce-Pr, I, Ca, and Sr, are listed. Zr and Nb were separable only when they were developed with mandelic acid of pH 5.2 and 7.9 Elements of the Ce group seemed to be separated when developed with acetylacetone-BuOH.

Kikuchi, Takehiko, and others. The metabolism of fission products. I. The metabolism of the radioactive ashes obtained from the No. 5 Fukuryu Maru: pp. 75–83.

When the radioactive ashes were administered by mouth, the radioisotopes which were chiefly absorbed were alkaline earths, and were deposited mainly in the bones. When, after the removal of the alkaline earths, the radioisotopes contained in the radioactive ashes were administered by mouth in the form of chloride or citrate, the radioisotopes chiefly absorbed were heavy metals such as Ru and Rh.

———. I. Metabolism of the radioisotopes contained in the radioactive ashes obtained from the No. 5 Fukuryu Maru: pp. 84–90.

Among the radioisotopes obtained by separation from ashes on the ship, i. e., Y^{91}, $Ce^{141, 144}$, Pr^{144}, Ca^{45}, $Sr^{89, 90}$, $Ru^{103, 106}$, Rh^{106}, Zr^{95}, Nb^{95}, and I^{131}, Sr, Ca, and Y were accumulated chiefly in the bones of adult mice, and the elimination of radio-Sr from there was very slow. When administered by mouth, radio-Sr and Radio-Ca were readily absorbed from the digestive tract, while the absorption of radio-Y from the tract was poor.

———. Paper XI, Studies on the metabolism of fission products III. Radioautographic studies on the localization of radiostrontium and radiocalcium in the bones: pp. 99–105.

———. The effects of EDTA–Na (Na Ethylenediaminetetraacetate) upon the metabolism of radiostrontium and radioyttrium in mice: pp. 106–111.

The toxicity of EDTA–Na, inert Sr $(NO_3)_2$ and Ba $(NO_3)_2$ has been examined. Simultaneous injection of EDTA–Na showed no significant effect upon the distribution of radio-Sr in the bones of mice. The distribution of Radio-Y in the bones of mice tended to decrease following the simultaneous subcutaneous injection of Y^{91} and EDTA-Na.

Japanese Committee for Compilation of Report on Research in the Effects of Radioactivity. Research in the effects and influences of the nuclear bomb test explosions. Tokyo, Japan Society for the Promotion of Science, 1956: 1824 pp. in 2 v.

Tokyo, Japan. University. Fisheries Institute and Faculty of Agriculture. Studies on influence of radio-activity from the point of fisheries science. Tokyo, November 10, 1954, 215 pp.

Data for the Committee on the Effects of Radioactivity, Science Council of Japan. Preliminary report iv, by cooperation of staff under Profs. Mori, Matsue, Suehiro, and Hiyama.

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Canada. Defense Research Board. Fallout from large nuclear explosions with application to civil defense, by G. H. Gilbert. Ottawa, Canada, 1955, 10 pp. (Operational Research Group Memorandum No. 5516).

Appendix A: A report by the United States Atomic Energy Commission on the effects of high-yield nuclear explosions, 8 pp.

"It is important to note that information relating to high level winds is very sparse, and that no information is available on the distribution of particle size and radio-activity within the nuclear cloud resulting from high yield explosions. Any conclusions on results presented in this paper must therefore be considered tentative."

Great Britain. Atomic Energy Research Establishment. Aspects of the travel and deposition of aerosol and vapour clouds, by A. C. Chamberlain. Harwell, Berkshire, England, 1953, 35 pp. (A. E. R. E. HP/R. 1261.)

———. Determination of strontium in sea water using both radioactive and stable isotopes, by R. W. Hummel and A. A. Smales. Harwell, Berkshire, England, 1955, 5 pp. (A. E. R. E. C/R 1755.)

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———. The effects of Operation Hurricane on plants and soils, by R. Scott Russell [and others]. Harwell, Berkshire,England, 1955, 106 pp. (Soil-Plant-Animal Relationships Report 3).

Fallout samples collected in air filters by aircraft and on gage sheets 24 hours following the Monte Bello burst were analyzed.

———. The metabolism of strontium in man, by G. E. Harrison [and others]. Harwell, Berkshire, England, 1955, 18 pp. (Soil-Plant-Animal Relationships Report 2).

———. The Monte Bello rat, November 1953, by D. W. H. Barnes [and others]. Harwell, Berkshire, England, 1955, 22 pp. (Soil-Plant-Animal Relationships Report 1).

Rats inhabiting Monte Bello at the time of the atomic explosion were examined for both external signs of radiation damage and damage from the ingestion of contaminated food.

———. The radiological dose to persons in the United Kingdom due to debris from nuclear test explosions, by N. G. Stewart [and others]. Harwell, Berkshire, England, 1955, 20 pp. (A. E. R. E. HP/R 1701).

———. The radiological dose to persons in the United Kingdom due to debris from nuclear test explosions prior to January 1956, by N. G. Stewart [and others]. Harwell, Berkshire, England, 1956, 22 pp. (A. E. R. E. HP/R 2017).

———. Radiostrontium and radiocaesium measurement in biological material, to December 1956, 11 pp. (A. E. R. E. HP/R–2182).

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Methods are described for the determination of strontium 90 due to fallout from nuclear explosions in soils, vegetation, bones, and milk. Current levels in Britain are shown to be very similar to those reported for United States.

———. Some factors influencing the gastro-intestinal absorption of strontium in rats, by H. G. Jones. Harwell, Berkshire, England, 1955, 3 pp. (Soil-Plant-Animal Relationships Report 6).

———. Summary of leading points [of Tripartite Conference on Soil-Plant-Animal Relationships of Fission Products]. Harwell, Berkshire, England, 1955, 2 pp. (Soil-Plant-Animal Relationships Report 15.)

"Order of importance of hazards from fallout in initial period is (*a*) external radiation, including beta radiation from activity on skin, (*b*) contamination of herbage and crops, (*c*) inhalation of activity and contamination of water supplies. With passage of time (*b*) becomes dominant."

Great Britain Atomic Weapons Research Establishment. Agricultural and biological investigations pertaining to contamination by fission products. Aldermaston, Berkshire, England, July 16, 1954, 26 pp.

Great Britain Medical Research Council. The hazards to man of nuclear and allied radiations. London, Her Majesty's Stationery Office, 1956, 128 pp.

Concludes that risk of radiation is controllable within limits that man can accept.

INDIAN

India (Republic) Defense Science Organisation. Nuclear explosions and their effects. Foreword by Jawaharlal Nehru. [Delhi] Publications Division, Ministry of Information and Broadcasting, Government of India, August 1956, 184 pp.

Bibliographical footnotes.

Scholarly analysis of the strontium hazard fills about 10 percent of the text. A three-page chapter contains a warning for the future and shows the great concern of India about the continued nuclear tests.

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U. S. S. R. Academy of Sciences. Conference on the peaceful uses of atomic energy. July 1–5, 1955. Session of the Division of Biological Science. Washington, United States Atomic Energy Commission, 1956, 198 pp., in English translation by Consultants Bureau.

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Morse, Wayne. Address . . . Radiation hazards. Remarks in the Senate. Congressional Record [daily edition] v. 103, May 23, 1957: 6747–6750.

Senator Morse discusses the unfortunate effects of radiation on the human system, particularly on the reproductive processes, and berates the administration for its failure to control or stop the testing of atomic weapons.

National Radiation Health Institute to protect mankind from atomic fallout. Congressional Record [daily edition], v. 103, February 14, 1957: 1763–1769

Bill introduced by Senator Neuberger to create a National Radiation Health Institute to perform research in effects of radiation on health. Contains reprints of Unna, Warren. Fallout held sure to hurt 6,000 babies. Washington Post.

Payne, Frederick G. United States participation in scientific commission within the United Nations to study certain effects of nuclear explosions. Congressional Record, v. 101, April 13, 1955: 4339–4340.

United States Atomic Energy Commission. Division of Biology and Medicine. Report on questions raised by Mrs. Jan Howard, East Palo Alto, Calif., regarding radioactive fallout from weapons tests. Extension of remarks of Hon. Charles S. Gubser. Congressional Record, v. 101, May 25, 1955: 7075–7076.

United States Congress. House. Committee on Appropriations. Fallout from nuclear explosions. Testimony of witnesses from Armed Forces Special Weapons Project, Army and Navy. In Department of Defense appropriations for 1958; hearings before the subcommittee of the Committee on Appropriations, 85th Congress, 1st session. Washington, United States Government Printing Office, 1957: pt. 2, p. 1532.

Contains reprint of Kulp, Laurence J. and others. Strontium-90 in man.

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Statements by Willard F. Libby: pp.4–66; Willard Bascom: pp. 127–190; Merle A. Tuve: pp. 191–211; Lester Machta: pp. 601–634; Lauriston S. Taylor: pp. 725–756; Ralph E. Lapp: pp. 763–797; Charles L. Dunham: pp. 899–925; Eugene P. Cronkhite: pp. 925–938; H. Bentley Glass: pp. 2709–2742.

——— ———. Civil defense for national survival. Twenty-fourth intermediate report of the Committee on Government Operations. Washington, United States Government Printing Office, 1956, 103 pp. (House Report No. 2946).

Nuclear explosion effects: pp. 8–9.

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Testimony by various authorities, including AEC and Weather Bureau experts, on various phases of possible hazards from atomic tests. Eisenbud states the cumulative fallout in the United States from early 1951 to January 1, 1955, varies from 21 millicuries per square mile in Arizona to 120 millicuries per square mile in New Mexico. The normal radioactive background in the United States varies from about 0.01 to 0.05 mr/hr. On six occasions, the background radiation was elevated to about 1 mr/hr beyond a few hundred miles from the test site, Troy, Chicago, Rochester, Salt Lake City (twice) and one other.

——— ———. Hearings on atomic-hydrogen weapon radiation, Congressional Record [daily edition], v. 103, May 22, 1957: 6659–6664.

List of witnesses and a detailed outline describing the scope and content of the hearings.

——— ———. Senate. Committee on Armed Services. Testimony [on fallout] of Gen. James M. Gavin, Chief of Research and Development, Department of the Army, in study of airpower; hearings before the Subcommittee on the Air Force (Symington Committee) of the Committee on Armed Services, 84th Congress, 2d session, Washington, United States Government Printing Office, 1956: pt. X, pp. 860–861.

——— ———. Senate. Committee on Armed Services. Civil defense program; hearings before the Subcommittee on Civil Defense * * * 84th Congress, 1st session, February 22, 26, March 3, 4, and 8, 1955. Washington, United States Government Printing Office, 1955, 920 pp. (in 2 pts.) (committee print).

Statement of Willard F. Libby: pp. 4–22.

Exhibit 1. Statement [on fallout] by Lewis L. Strauss: pp. 231–233.

A report by the United States Atomic Energy Commission on the effects of high-yield nuclear explosions: pp. 234–240.

Exhibit 2. Remarks by Dr. Willard F. Libby for delivery at the Washington Conference of Mayors, Washington, D. C., Thursday, December 2, 1954: pp. 240–244.

Exhibit 3. Atomic test effects in the Nevada Test Site Region: pp. 244–253.

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Statements by Mrs. Adelaide N. Baker: pp. 478–492; Arthur H. Compton: p. 819; Mrs. George Gellhorn: pp. 900–903; T. Alexander Pond: pp. 907–911; Mrs. Frederick Faust: pp. 951–953; Warren Weaver: pp. 1135–1154.

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Vol. 7: Nuclear chemistry and effects of irradiation:

Paper 1058: Radiochemical analysis of radioactive dusts, by Kenjiro Kimura (Japan): pp. 196–209.

Paper 1059: Radiochemical interpretation of the radioactive fallout, by Kenjiro Kimura and others (Japan): pp. 210–213.

Vol. 11: Biological effects of radiation:

Paper 392: The experimental animal for study of the biological effects of radiation, by J. F. Loutit (UK): pp. 3–6.

Paper 616: The influence of ionizing radiations on animal organisms, by A. V. Lebedinsky (U. S. S. R.): pp. 7–24.

Paper 803: Biological effects of radiation, by John C. Bugher (USA): pp. 45–48.

Paper 852: Studies on the radium content of humans arising from the natural radium of their environment, by A. F. Stehney and H. F. Lucas (USA): pp. 49–54.

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Paper 244: A formulation of the relation between radiation dose and shortening of life span, by H. A. Blair (USA): pp. 118–120.

Paper 1045: Radiation injury due to radioactive fallout, by Masao Tsuzuki (Japan): pp. 132–133.

Paper 88: The deposition of radioactive substances in bone, by Frank E. Hoecker (USA): pp. 138–145.

Paper 899: Biological damage resulting from exposure to ionizing radiation, by L. H. Gray (UK): pp. 209–212.

Paper 449: The genetic problem of irradiated human populations, by T. C. Carter (UK): pp. 384–386.

Paper 234: How radiation changes the genetic constitution, by H. J. Muller (USA): pp. 387–399.

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Paper 778: The general problems of protection against radiations from the public health point of view, by World Health Organization: pp. 10–14.

Paper 778: The achievement of radiation protection by legislative and other means, by Lauriston Taylor (USA): pp. 14–21.

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Paper 572: Radiation from clouds of reactor debris, by J. Z. Holland: pp. 110–118.

Paper 451: Radiation injury and protection—maximum permissible exposure standards, by W. Binks (UK): pp. 129–131.

Paper 89: Maximum permissible exposure standards, by Robert S. Stone (USA): pp. 132–138.

Paper 79: Maximum permissible concentration of radioisotopes in air and water for short exposure periods, by Karl Z. Morgan and others: (USA): pp. 139–158.

Paper 247: Permissible exposure to ionization radiation, by Lauriston S. Taylor (USA): pp. 196–197.

Paper 1041: Maximum permissible exposure standards, by Masanori Nakaidzumi (Japan): p. 198.

Paper 944: On the maximum permissible dose of X- and gamma-radiation, by W. Jasinski and I. Zlotowsky (Poland): pp. 199–200.

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Paper 276: Atomic energy and meteorology, by H. Wexler and others (USA): pp. 333–344.

Paper 1055: Radioactivity in rainwater and the air observed in Japan, 1954–1955, by Yasuo Miyake (Japan): pp. 345–349.

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Paper 393: The behavior of I^{131}, Sr^{89} and Sr^{90} in certain agricultural food chains, by A. C. Chamberlain and others (UK): pp. 360–363.

Paper 280: The accumulation of radioactive substances in aquatic forms, by R. F. Foster and J. J. Davis (USA): pp. 364–367.

Paper 1052: Biological cycle of fission products considered from viewpoint of contamination of marine organisms, by Yoshio Hiyama (Japan): pp. 368–370.

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Paper 281: Radioactivity in terrestrial animals near an atomic energy site, by W. C. Hanson and H. A. Kornberg (USA): pp. 385–388.

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United States and United Kingdom exchange views at Bermuda meeting. Department of State bulletin, v. 36, April 8, 1957: 561–562.

Annex II deals with nuclear tests and methods of limitation.

Wadsworth, James J. Coordination and dissemination of information on effects of atomic radiation. Department of State bulletin, v. 33, November 21, 1955: 851–858.

Statements of United States Representative to the General Assembly pointing out the need for a fact gathering service to coordinate and disseminate information about the dangers of radioactivity.

Warren, Shields. Launching the U. N. study of effects of atomic radiation. Department of State bulletin, v. 34, May 21, 1956: 860–861.

First meeting of Scientific Committee on Effects of Atomic Radiation, March 14–23.

APPENDIX 10

OAK RIDGE NATIONAL LABORATORY,
Oak Ridge, Tenn., August 21, 1957.

Mr. JAMES T. RAMEY,
Executive Director, Joint Committee on Atomic Energy,
Washington, D. C.

DEAR MR. RAMEY: Enclosed please find a copy of the material concerning topic VIII D of the outline, fallout and water decontamination, requested by Congressman Holifield for the Joint Committee on Atomic Energy Report.

Enclosed is the biographical sketch also requested in your letter of June 19, 1957.

If I can be of any further assistance to you and the committee, please feel free to write.

Thank you.

Very truly yours,

WILLIAM J. LACY,
ERDL Representative at ORNL.

Enclosures: 1. Report on Fallout. 2. Biographical sketch.

Cc: Commanding Officer, Engineer Research and Development Labs, Fort Belvoir, Virginia; Harry N. Lowe, Jr., Chief Sanitary Engineering Branch, Fort Belvoir, Virginia; Dr. Karl Z. Morgan, Director, Health Physics Division, Oak Ridge National Laboratory, Oak Ridge, Tennessee.

BIOGRAPHICAL SKETCH

William J. Lacy was born in 1928 in Wallingford, Connecticut, attended Lyman Hall High School where he won the prizes in science and chemistry, then he obtained a B. S. degree in 1950 from the University of Connecticut where he majored in Chemistry. He entered graduate school at New York University in September of 1950 and worked as a research associate on an AEC research contract. In May of 1951 he joined the staff at the Engineer Research and Development Labs of Fort Belvoir, Virginia, and immediately was transferred to the Oak Ridge National Laboratory to work on the water decontamination research project.

He has had seven (7) articles published, presented numerous papers and is a member of the American Chemical Society, American Association for the Advancement of Science and the Scientific Research Society of America.

Mr. Lacy is married and has two (2) sons, 2½ and six months, he resides in Oak Ridge, Tennessee.

[Material for Joint Committee on Atomic Energy Topic VIII D]

REMOVAL OF RADIOACTIVE FALLOUT FROM CONTAMINATED WATER SUPPLIES

William J. Lacy, Chemist,* Sanitary Engineering Branch, Engineer Research and Development Labs, Fort Belvoir, Va.

There are two possible sources of radioactive contamination of public water supplies, (1) the result of direct discharge into the environment from reactor processing plants, research center using radioisotopes and others and (2) deposition of radioactive material by fallout or wash-in due to weapon's test activities.

Most of the radioactive materials in item one are in solution, fallout, however, may be in the form of insoluble oxides, and its removal may differ from the removal of ionic material.

Studies have been reported on the subject of fallout in particular areas (1), (2), (3), (4). It was reported that 35 percent of the fallout activity was removed by the Albany, New York, water treatment plant, an alum coagulation, settling and filtration plant. Thomas and his coworkers at Harvard (2) (3) working at the Lawrence, Massachusetts, water plant obtained 80 percent removal by coagulation, settling, and filtration. Bell (4) compared the fallout removal results from Cambridge and Lawrence, Massachusetts, and Rochester, New York, with pilot plant results obtained by Straub (5) (6) who used a simulated bomb blast mixture with an age about one month after detonation.

*On loan to Health Physics Division, Oak Ridge National Lab., Oak Ridge, Tennessee.

The comparison indicated the three treatment plants show much lower removals of fallout than Straub obtained on chemical processed radioactive material even though the same procedure was used in both cases. The U. S. P. H. S. reported the analysis of rain water samples containing fallout showed 50 to 100 percent of the "old" radioactive material to be soluble. However, the soluble fraction dropped to about 30 percent during the weapon's testing period.

For reactor made fission products, or a mixture of commercially available radioisotopes, the efficiency of removal would be a function of the various radioelements comprising the mixture. Results in laboratory studies and pilot plant scale investigations by the author indicates removals of about 70 to 85 percent using either alum and soda ash or ferric chloride and limestone coagulants. A series of studies (7) reported that removals of 99 percent could be obtained using a serial coagualtion procedure including an excess lime-soda ash softening or phosphate coagulation step, provided some clay material was added to remove radiocesium.

Conventional wastes treatment processes include coagulation, settling, and filtration, plus disinfection. Often additional treatment, such as fluoridation, aeration, softening, ion exchange, iron and manganese removal are employed.

During coagulation certain of the dissolved constituents are precipitated as insoluble hydroxides or carried along, scavenged, with the heavy metal hydroxides of alum or iron. Coagulation can have its radioactivity removal increased from about 75 percent to almost 90 percent by the addition of clay for cesium and copper sulfate for radioiodine.

It should be pointed out that different radioisotopes respond differently to removal by coagulation. Other factors to be considered include: (1) Chemical and physical form of the radionuclide, (2) concentration or the radioactive material, and (3) optimum pH of flocculation for the coagulant available and the water under treatment. Investigation by the author (8) indicates increase dosages of chemical generally yielded only slightly higher removals while higher pH usually resulted in proportionately higher removals.

Softening using lime-soda ash is one of the more effective chemical methods for the removal of radiostrontium and barium. However, it is necessary to use excesses quantities, over the stoichiometric dosage, for satisfactory results. Studies at MIT (9) (10) have indicated that the radiostrontium is removed by coprecipitation with the hardness or calcium carbonate in a mixed crystal formation.

Ion exchange is another method used by some municipal water treatment plants. Removal of ionic radionuclides by this process is not only technically possible (11), but very satisfactory. The most effective method employes either a mixed bed principal or separate cation-anion exchange columns. Ion exchange units such as home-type water softeners are very effective for removal of 99+ percent of the radioactive fallout or reactor originated radionuclides from contaminated water. Also ion exchange resins (mixed) can be used with, good results, as slurries for the removal of a vaniety of radioactive contaminants from water solutions (12)

Other methods, such as, the use of clays, powdered metal, charcoal, flotation and various adsorbents all have some merit for the removal of specific radioisotopes or under a given set of condition result in good removals. (13) However, clay seems to have the most practical and over advantage of being (1) available, (2) cheap, (3) effective, (4) simple to use, (5) easy to remove both absorbent and absorber and the radioactive material will not be easily leached once it is attached to the clay particle. Distillation although not a usual municipal water treatment method is used extensively by the military on island bases and where a high quality of water is required. Distillation results in the best single treatment of a contaminated water removing 99.9+ percent. (14) The major objection to distillation as a water treatment procedure is cost.

As indicated by the literature cited most of the above studies have been made on chemically processed, radiochemically pure radioisotopes and not true fallout from a nuclear detonation. Therefore, it was expected that the actual fallout material not being entirely in the same physical and chemical form could not be as readily removed from contaminated water. However, recent tests by the Corps of Engineers at the AEC Nevada Proving Grounds on some very low level fallout indicated (1) close agreement with laboratory results on removal by coagulation and softening using lime-soda ash and precipitation with trisodium phosphate at a high pH, (2) the ion exchange procedures resulted in 99 to 100 percent removal of the bomb fallout material, (3) the material that was not

a true solution could be removed physically and the material in solution treated chemically and (4) radionuclide once adsorbed on clays were not appreciably leached by tap water.

Many other experiments have been made by myself and others, some are still in progress, which have not been cited here. It is felt that this brief general review plus the six tables showing detailed data, will give the committee a review of the field on water decontamination.

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TABLE I.—*Coagulation for removal of radioactivity*

Contaminant	Dosage, p. p. m.	Percent removal	
		$FeCl_3$-$CoCO_3$	Alum-soda ash
Ce^{144}-Pr^{144}	50	99.2	96.1
	100	99.4	96.5
Ba^{140}-La^{140}	50	67.4	58.4
	100	70.7	58.0
Zr^{95}-Nb^{95}	50	98.1	76.4
	100	98.8	78.6
I^{131}	50	45.0	26.3
	100	63.0	35.7
P^{32}	45.7	93.3	94.1
MFP-1 [1]	29–58	60–83.7	
MFP-2 [2]	50	70.1	72.6

[1] MFP-1—ORNL waste containing mixed fission products.
[2] MFP-2—Simulated 30-day atomic-bomb blast mixture.

TABLE II.—*Results of lime-soda ash treatment for removal of strontium*

Treatment	Percent removal of activity
Stoichiometric amounts	75.0
20 ppm excess lime-soda ash	77.0
50 ppm excess lime-soda ash	80.1
100 ppm excess lime-soda ash	85.3
150 ppm excess lime-soda ash	97.3
200 ppm excess lime-soda ash	99.4
300 ppm excess lime-soda ash	99.7

TABLE III.—*Ion exchange column for water decontamination*

Run No.	Resin*	Contaminant	Resin capacity gal./ft.^{3}	Percent removal until breakthrough
1	Cation	MFP-1	5,700	71–82
2	Mixed bed	MFP-1	3,150	93–99+
3	Cation	MFP-2	6,000	88–96
4	Mixed bed	MFP-2	2,890	96–99
5	Cation	Zr^{95}–Nb^{95}	6,750	85–88
6	Mixed bed	Zr^{95}–Nb^{95}	2,600	92–97
7	Cation	MFP-3	3,270	85–90
8	Mixed bed	MFP-3	6,150	92–99

*Cation resin was a high capacity nuclear sulfonic acid type and mixed bed was amberlite MB–3.

NOTES

MFP-1—ORNL liquid waste material.
MFP-2—Simulated 30-day atomic-bomb debris.
MFP-3—Three year old dissolved reactor fuel element.

TABLE IV.—*Removal of radioactive contaminants from water—Resin-jar test studies (stirring time, 90 minutes, samples filtered)*

Contaminant	Initial pH	Initial activity c/m/ml	Percent removal mixed ion exchange resin, p. p. m.			
			450	900	1,800	2,700
P^{32}	8.2	5,560	47.4	74.5	96.2	99.8
Cd^{115}	8.0	7,680	37.9	45.6	91.1	99.99
Cs^{137}–Ba^{137}	8.2	8,200	15.1	14.6	69.1	99.99
Zr^{95}–Nb^{95}	8.1	6,700	98.3	98.4	99.2	99.4
I^{131}	7.5	3,260	84.5	93.5	95.6	98.1
$Ce^{141, 144}$–Pr^{144}	7.9	4,150	98.7	99.2	99.8	99.98
Ba^{140}–La^{140}	7.6	3,490	85.1	94.5	96.8	99.9
FPM-4	8.3	13,600	82.7	90.5	97.3	99.2
FPM-5	2.7	8,400	38.4	----	----	----

NOTES

FPM-4—Iodine dissolver solution aged 30 days.
FPM-5—Mixed fission product waste containing mainly Cs^{137}–Ba^{137} and Ru^{106}–Rh^{106}.

TABLE V.—*Decontamination of radioactively contaminated water by slurrying with clay*

Contaminant	pH	Clay concentration, p. p. m.	
		1,000	3,000
		Percent removal	
Ru^{106}-Rh^{106}	5.2	50.5	61.5
Zr^{95}-Nb^{95}	7.5	98.0	99.4
Sr^{90}-Y^{90}	7.7	83.4	92.9
I^{131}	7.5	4.9	3.4
$Ce^{141,144}$-Pr^{144}	8.0	99.7	99.9
Ba^{140}-La^{140}	7.8	88.8	94.3
MFP-1	8.8	82.0	86.3
MFP-2	9.0	70.0	72.8
MFP-3	7.7	79.0	83.6

TABLE VI.—*Removal of radioactive material by distillation (60 gallon/hr thermocompression unit)*

Run No.	Contaminant	Activity of feed, d/m/ml	Removal of activity expressed as decontamination factor	Percent
1	MFP-1	22,060	4.10×10^3	99.98
2	MFP-2	97,400	4.97×10^3	99.98
3	MFP-3	31,150	3.59×10^3	99.97
4	MFP-4	62,400	3.52×10^3	99.72
5	Pa^{233}	41,030	2.31×10^3	99.96
6	I^{131}	60,900	7.04×10^3	99.86
7*	MFP-5	38,910	1.09×10^3	99.91
8*	MFP-4	69,700	1.00×10^4	99.99
9*	MFP-1	12,020	1.70×10^4	99.99
10*	I^{131}	45,600	1.28×10^3	99.92
11*	Pa^{233}	25,300	5.80×10^3	99.98

*Glass wool reflux condenser used.

NOTES

MFP-1 was 3-year-old fission product mixture.
MFP-2 was a 2-week-old mixture from disolution of a reactor slug.
MFP-3 was composite sample or ORNL liquid waste.
MFP-4 concentrate from ORNL liquid waste evaporator.
MFP-5 mixture to simulate the material expected 10 days after atomic detonation.

APPENDIX 11

UNITED STATES ATOMIC ENERGY COMMISSION,
Washington 25, D. C., August 20, 1957.

Hon. CHET HOLIFIELD,
Chairman, Special Subcommittee on Radiation of the Joint Committee on Atomic Energy, House of Representatives, Congress of the United States.

DEAR MR. HOLIFIELD: At the suggestion of your Committee, the Division of Biology and Medicine, U. S. Atomic Energy Commission, invited the principal participants in the discussions involving predictions of future skeletal concentrations of strontium 90 in humans which took place at the recent Congressional Hearings on fallout to meet once again in an attempt, insofar as present information permitted, to reduce the degrees of uncertainty in these predictions.

This meeting took place on July 29, 1957 and I am pleased to transmit a summary report of the meeting based on the stenographic transcript and consultation with the principal participants. This report was prepared by Dr. Forrest Western, of the Division of Biology and Medicine. It is my opinion this report honestly and clearly reflects the views of the participant scientists with respect to this problem. This document, then, would appear to reflect the thinking of those scientists who have worked hardest and thought most on the subject of these predictions, and should, therefore, be a useful addition to the text of the very important and

informative Hearings which your Committee held in May and June of this year on the whole matter of fallout from weapons tests.

Sincerely yours,

CHARLES L. DUNHAM, M. D.,
Director, Division of Biology and Medicine.

PREDICTED SKELETAL CONCENTRATIONS OF STRONTIUM 90

INFORMAL DISCUSSION OF JULY 29, 1957

At the suggestion of the Joint Committee on Atomic Energy, the Division of Biology and Medicine, U. S. Atomic Energy Commission, invited the principal participants in discussion of this subject at the recent Congressional Hearings, May 27–June 6, 1957, to meet in Washington, D. C., July 29, to try to reduce the degrees of uncertainty involved in various predictions of future skeletal concentrations of strontium 90 in humans. These persons were: Mr. Merril Eisenbud, Dr. J. Laurence Kulp, Dr. Wright H. Langham, Dr. Willard F. Libby, Dr. Lester Machta, Dr. William F. Neuman and Dr. Walter Selove. In addition to these participants were: Dr. Charles L. Dunham who alternated with Dr. Libby as Chairman of the discussion, Dr. Lyle T. Alexander, Mr. Hal Hollister, Dr. J. Calvin Potts, Dr. Robert Reitemeier, and the author of this summary, Forrest Western. This summary is based on a stenographic transcript and has had the benefit of comments by most of the participants.

It was generally agreed that the extensive measurements by Dr. Kulp of concentrations of strontium 90 in human skeletons established one base from which one may extrapolate skeletal concentrations of strontium 90 to be expected in the future. Dr. Kulp stated that the average concentration in children of the northeastern United States in the fall of 1956 was about 0.8 micromicrocuries of strontium 90 per gram of calcium. In discussion of relationships between concentrations in the United States and other parts of the world, Dr. Kulp indicated that concentrations in children of northeastern United States are consistent with those of two widely separated areas in other parts of the world from which he has been able to obtain a considerable number of samples. Dr. Selove discussed reports of relatively high local concentrations of fallout in an Asiatic area and suggested that possible fluctuations in local patterns of tropospheric fallout in the periods shortly following tests explosions might result in considerably higher skeleton concentrations in some areas of the world. Although our world sampling program to date has failed to disclose areas in which skeletal concentrations are higher than in the United States, individual comments endorsed the desirability of continued search for such areas as a part of our world-wide study of the distribution and uptake of fallout from nuclear detonations.

It was agreed that, *even if fallout had ceased at the end of 1956*, skeletal concentrations of strontium 90 would be expected to increase until they came into equilibrium with the strontium 90 in the environment. Somewhat independent estimates of the equilibrium value may be made (1) from a knowledge of *changes* in environmental (specifically, *dietary*) concentrations during the growth of the skeletons assayed, and (2) by application of factors of discrimination between calcium and strontium in estimating the uptake of strontium 90 from existing concentrations of strontium 90 (a) the soil, (b) the over-all diet, or (c) milk. Each of these methods involves some degree of uncertainty. After discussion of the uncertainties involved, it was agreed that these various considerations make it appear probable that average skeletal concentrations to be expected in young persons of northeastern United States as a result of strontium 90 actually deposited on the earth's surface up to the end of 1956, fall between 1.5 and 2 micromicrocuries per gram of calcium.

Because some of the strontium 90 released to the stratosphere in past years has not yet been deposited on the earth's surface, actual skeletal concentrations from tests performed before the end of 1956 may be expected to become greater than the values estimated under the conditions assumed above. The increase in concentration to be expected from additional fallout of this material depends upon a number of factors; (1) the additional activity reaching the surface of the earth, (2) the fact of delay in the appearance of additional strontium in the diet, (3) the relative importance of total soil content and rate of fallout (specifically, rate of retention on surfaces of vegetation), (4) radioactive decay and (5) decrease, with time after fallout, in the percentage of strontium 90 in the soil which is available for uptake by plants. It appeared from the discussion that the greatest uncertainty involved here is in predicting the distribution and time of fallout to be expected from residual stratospheric content.

It was estimated that, in addition to the range of uncertainty by a factor of 1.25 represented in the above estimate of the average skeletal concentrations to be expected from strontium 90 actually deposited on the earth's surface by the fall of 1956, there might be an additional range of uncertainty, by a factor as large as two, in estimates involving the ratio of the quantity of strontium 90 in the stratosphere to that on the ground. In estimating total skeletal concentrations in the northeastern United States which might be expected when fallout of strontium 90 produced prior to 1957 is essentially complete, these two uncertainties alone would result in a range of a factor of 2.5 between minimum and maximum estimates.

It was agreed that the first effort of the group would be to estimate average skeletal concentrations to be expected in the age group of maximum concentration in 1975, assuming that there were no nuclear detonations after 1956. Dr. Kulp estimated that, because of radioactive decay and decrease in availability, additional fallout of strontium 90 produced before 1957 would not make environmental concentrations of strontium 90 in the northeastern United States in 1957 significantly greater than in 1956. After discussion, this led to the estimate that, if the residual stratospheric content were to be deposited with uniform distribution over the surface of the earth, skeletal concentrations in the age group of maximum concentration in 1975 would fall in the range of from 1.5 to 3.5 micromicrocuries of strontium 90 per gram of calcium.

Mr. Eisenbud, taking a different approach, first estimated quantities of strontium 90 on the surface in the northeastern United States at the end of 1956 due to tropospheric and stratospheric fallout, respectively, and the fraction of strontium 90 previously injected into the stratosphere which had fallen out by the end of 1956. Maximum future environmental levels were then related to those of 1956, using the assumption that fallout of the residual (1956) stratospheric content would have the same geographic distribution as the previous stratospheric fallout. Discussion of this approach led to an estimate of skeletal concentrations, in 1975, in the range from 2 to 5 micromicrocuries of strontium 90 per gram of calcium.

Dr. Machta discussed the possibility that strontium 90 injected into the stratosphere near the equator may be moving northward and entering the troposphere preferentially above 30° N., in such a manner that the fraction of the stratospheric content falling out in these latitudes is increasing with time. It was estimated that such "banding" of fallout might result in skeletal concentrations two times as high as those estimated on the basis of Mr. Eisenbud's assumption of a constant stratospheric fallout pattern; i. e., an average skeletal concentration in children of from 4 to 10 micromicrocuries per gram of calcium.

It was agreed that at this time our knowledge of atmospheric transport is too limited to reduce the range of uncertainty on this point represented by the ranges of estimated concentrations described in the preceding three paragraphs.

It was estimated that, if testing of weapons were continued up to about 1965 in such a manner as to produce strontium 90 in the same total quantity and with the same distribution (i. e., with tests held at the same geographical locations and with the same distributions of tropospheric and stratospheric fallout) as were produced prior to 1957, the average total skeletal content in young persons in about 1975 might be approximately 2.5 times that to be expected if there were no further testing. This factor is considered to apply equally to the three estimates discussed in the preceding paragraphs.

The estimates described above are summarized in the following table:

Predicted average skeletal levels of strontium to be expected in young persons in northeastern United States under various conditions of testing of nuclear weapons

[All levels are in micromicrocuries of strontium 90 per gram of calcium]

Concentrations in children, fall 1956	Predicted future concentrations from strontium 90 on ground before 1957	Predicted concentrations in 1975 from all strontium 90 produced before 1957	Predicted concentrations in 1975 if past tests or equivalent were repeated before 1965
0.8	1.5 to 2	1.5 to 3.5*	3.5 to 9.*
		2 to 5	5 to 12.
		4 to 10	10 to 25.

*The considerations leading to each of these 3 estimates are discussed in the text of this summary.

It was the consensus of the group that within one or two years the confidence with which predictions of future concentrations can be made will be so greatly increased that it is unprofitable for the present purpose to extend predictions beyond those given in the above table.

APPENDIX 12

UNITED STATES ATOMIC ENERGY COMMISSION,
Washington 25, D. C., June 1, 1957.

Hon. CHET HOLIFIELD,
Chairman, Special Subcommittee on Radiation, Joint Committee on Atomic Energy, Congress of the United States.

DEAR MR. HOLIFIELD: Your letter of 31 May 1957 requests a letter on an unclassified basis giving: (a) an estimate as to year-to-year fission yield and total yield put into the atmosphere by atomic weapons from all sources; (b) a list of weapons test series conducted by each country; and (c) a listing of the number of explosions which have taken place, country by country and year by year.

As to total yield and total fission yield released by tests to date either in gross or by nation, this information has security implications which preclude my giving it as unclassified data. Various unclassified estimates which do not reveal such totals have been made as to the amount of fission debris making its way into the stratosphere. One such estimate is that given on pages 952 and 954 of the attached report, "Current Research Findings on Radioactive Fallout," by Dr. Willard F. Libby.

Information as to U. S. test series and the number of shots in each is contained in the following table:

Name of series	Date	Number of shots
Trinity	1945	1
Crossroads	1946	2
Sandstone	1948	3
Greenhouse	1951	4
Ranger	1951	5
Buster/Jangle	1951	7
Tumbler/Snapper	1952	8
Ivy	1952	2
Upshot/Knothole	1953	11
Castle	1954	(1)
Teapot	1955	14
Wigwam	1955	1
Redwing	1956	(1)
Plumbbob	1957	[2] 1

[1] The number of shots in Castle and Redwing is still classified.
[2] Only 1 shot to date.

The above list is exclusive of the two weapons detonated in combat during World War II.

With respect to the British series, the United Kingdom has announced the following series and number of shots:

Location	Date	Number of shots
Australia	1952	1
Do	1953	2
Do	1956	2
Do	1956	4
Christmas Island	1957	2

Of several Soviet references to their tests, they have: announced one on 20 August 1953, shortly before the U. S. announcement was issued; made a general statement on 18 September 1953 to the effect that they had conducted several tests in recent weeks which constituted confirmation of the U. S. announcement of 31 August 1953; and announced a test on 10 September 1956 about which the U. S.

made no comment. The United States has from time to time announced certain of the tests. Copies of these announcements are attached. From a review of these announcements it can be noted that the U. S. has announced 23 specific USSR detonations. A complete list of all detected shots cannot be provided on an unclassified basis. In this respect on August 26, 1956, the Chairman of the AEC stated: "Although there have been but 13 announcements by the U. S. regarding Soviet testing, several have noted a series of detonations and the actual number of Soviet detonations is significently higher than 13."

Should you desire a classified summarization of the information requested which cannot be given on an unclassified basis, I would be most pleased to provide this separately.

Sincerely yours,

K. E. FIELDS,
General Manager.

[From Congressional Record, June 18, 1957]

NUCLEAR WEAPONS EXPLOSIONS

The following table has been compiled principally from press releases of the United States Atomic Energy Commission. However, reports in the press as to the size and nature of various explosions have been included when available.

On April 12, the United States had announced 19 Soviet tests. The AEC has pointed out that this country does not disclose all of the U. S. S. R. shots of which it has knowledge but limits itself to statements about explosions of special interest. The actual number of Soviet detonations is therefore significantly higher than those announced.

As of June 17 the AEC had announced 68 tests by the United States. However, the total number of detonations made by this country has never been announced.

The United Kingdom has announced 11 tests to date, June 18, which is understood to be the total number of tests made by that country.

Year	United States		U. S. S. R.		United Kingdom		Remarks
	Date	Place	Date	Place	Date	Place	
1945	July 16	Alamogordo					1st test of A-bomb.
	Aug. 6	Hiroshima					Air burst, energy release about 20,000 tons of TNT.
	Aug. 9	Nagasaki					Do.
1946	July 1	Operation Crossroads, Bikini Lagoon.					Air burst, Nagasaki-type bomb.
	July 25						Underwater detonation.
1948 (spring)		Operation Sandstone, Eniwetok Atoll.					3 explosions announced May 17, no details given.
1949			Sept. 23	Soviet territory			1st atomic explosion in U. S. S. R. announced by President Truman.
1951 (winter)	Jan. 27	Operation Ranger, Nevada Flats.					5 tests primarily for tactical information, Air bursts.
	Jan. 28						
	Feb 1						
	Feb. 2						
	Feb. 6						
1951	April and May	Operation Greenhouse, Eniwetok.					4 tests described as "experiments contributing to thermonuclear research."
			Oct. 3	Soviet territory			2d explosion. Reported by the United States, later confirmed by the Kremlin.
			Oct. 22	do			President Truman announced evidence of a 3d nuclear explosion by the U. S. S. R.
1951	Oct. 22	Operation Buster Jangle, Nevada Flats.					7 air, tower, and surface or underground bursts. Low yield. Tactical.
	Oct. 28						
	Oct. 30						
	Nov. 1						
	Nov. 5						
	Nov. 19						
	Nov. 29						
1952	Spring and summer	Operation Tumbler Snapper, Nevada Flats.					8 air and tower bursts. Troop participation in some tests.
	Apr. 1						
	Apr. 15						
	Apr. 22						
	May 1						
	May						
	May 25						
	June 1						
	June 5						
	Fall	Operation Ivy, Eniwetok					2 tests. 1 was the 1st hydrogen bomb ever exploded. Yield 5 megatons.
					Oct. 3	Monte Bello Islands.	Nature of bomb not disclosed. Probably of Hiroshima type.

Year	United States		U. S. S. R.		United Kingdom		Remarks
	Date	Place	Date	Place	Date	Place	
1953	Mar. 17	Operation Upshot Knothole, Nevada Flats.					11 tests: Air, tower, and 1 shot from a 280-millimeter gun. The largest explosion, that of Apr. 18, was reportedly equal to 30,000 to 40,000 tons of TNT.
	Mar. 24						
	Mar. 31						
	Apr. 6						
	Apr. 11						
	Apr. 16						
	Apr. 25						
	May 8						
	May 19						
	May 29						
	June 4						
			Aug. 12	Soviet territory			Initial hydrogen bomb (nondeliverable).
			Aug. 31	do			Fission explosion in same range of energy release as Nevada Flats tests.
1954	Mar. 1	Operation Castle, Bikini					Hydrogen bomb reported to have had a yield of 15 megatons. Radioactive fallout over 7,000 square miles.
	Mar. 26						No information released concerning this test.
	Apr. 6	Eniwetok					3d test in the series. Presumably an H-bomb.
			Oct. 26	Soviet territory			Announcement made by the AEC of a series beginning in September.
1955	Feb. 18	Operation Teapot, Nevada Flats.					14 air, tower, and underground tests. Included atomic trigger for detonating H-bombs, also civil defense test and "air-killer" test.
	Feb. 22						
	Mar. 1						
	Mar. 7						
	Mar. 12						
	Mar. 22						
	Mar. 23						
	Mar. 29 (2 tests)						
	Apr. 6						
	Apr. 9						
	Apr. 15						
	May 5						
	May 15						
	May 17	Operation Wigwam, off west coast.					Small underwater atomic weapon exploded, probable yield 1,000 to 5,000 tons of TNT.
			Aug. 4	Soviet territory			AEC announced that the Soviets had resumed testing of nuclear weapons.
			Sept. 24	do			Announcement by AEC of another Soviet nuclear explosion.
					Oct. 15	Woomera Rocket Range, Australia.	2 tests, the 1st equivalent to 20,000 tons of TNT. No data for the 2d test.
			Nov. 10	Soviet territory			Announcement by AEC of a further test in the 1955 series.

			Nov. 23	do			Hydrogen bomb in the range of megatons. Khrushchev announced on Nov. 27 that it was a hydrogen bomb and that it had been dropped at a great height from an airplane.
1956			Mar. 21	Soviet territory, probably central Asia.			AEC announced another Soviet nuclear explosion.
			Apr. 2				Another explosion in current series announced by AEC.
	May 5	Operation Red Wing, Eniwetok.					Relatively small device exploded, about 10 kilotons.
					May 16	Monte Bello Islands.	Test expected to supply data for triggering of H-bomb.
	May 21	Bikini					1st H-bomb dropped by U. S. Air Force. Yield at least 15 megatons.
			Aug. 24	Southwestern Siberia.			
			Aug. 30				A series of small explosions.
			Sept. 2				
					Sept. 27	Maralinga Proving Ground, South Australia.	4 tests, tower and air; new and cheaper kinds of A-bombs tested.
			Nov. 17	Southwestern Siberia.			Small explosion.
			Jan. 19				
			Mar. 8				
			Apr. 3				
			Apr. 6	do			Moscow announced these tests were designed to perfect atomic warheads for tactical purposes.
			Apr. 10				
			Apr. 12				
			Apr. 16				
					May 15	Christmas Islands.	H-bomb dropped from Vikers Valiant jet bomber. Energy release 1,000,000 tons of TNT.
	May 28	Beginning of Operation Plum Bob, atomic test site, Nevada. The tests are scheduled to continue into September.					Probably a warhead for small rockets or missiles. Yield about 10,000 tons of TNT.
					May 31	Christmas Islands.	2d hydrogen bomb exploded by the British.

[1] 1 of a series.

Prepared for the use of the Subcommittee on Disarmament by Janie E. Mason, of the subcommittee staff, on loan from the Library of Congress, June 6, 1957.